Remarks

ON

THE PROBLEM OF THE CHILD IN THE
TUBERCULOUS HOUSEHOLD*

BY

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SURREY

Research on the problem of tuberculosis in childhood is a great feature of present-day activities. Facts and figures are pouring in from every civilized country. It might almost seem that the tuberculosis of adults was being neglected and that the best of our attention was being directed to childhood tuberculosis. I think this is in some degree true, and the reason for it is that there is a hazy idea in the air that in the prevention and treatment of childhood tuberculosis lies the key to the problem of the prevention of the disease in adults. This may or may not be true. It is, however, important to realize that the researches on tuberculosis in childhood are not finished; they are hardly started indeed, the results therefrom are as yet but partially digested, and the truths that they seem to show to-day may be profoundly modified as further knowledge is acquired.

The treatment and prevention of adult tuberculosis has been undertaken as a national duty, and Sir Robert Philip, one of the fathers of the movement, in his Malcolm Morris Memorial Lecture claims that the rapid decrease in the mortality from the disease may be ascribed to the work of this movement. His statement may meet with criticism, but no one can deny that the decrease has at any rate accelerated with the movement. Such a national duty involves the tax-payer, through the State, in great expense, and if it is to be enlarged and intensified in the direction of our childhood population, as Sir Robert Philip suggests, this expense must increase.

In addition to the financial burden public health regulations will follow, and laws which are bound to affect the liberties and perhaps the happiness of the individual, both child and adult. Neither financial responsibility nor wise law-giving must be shirked if these are going to be of real benefit to the community, but they must be founded upon real truths and not upon hasty conclusion. In the meanwhile, before any national movement is made, if it is made at all, the general practitioner and the public health official are still day by day confronted with the problem of the child in the tuberculous household; and from the way in which this problem is being tackled, or ignored, in different countries and in different parts of the same country, it would seem that the profession is wandering in the dusk of a thick forest of conflicting opinions, along ill-made tracks where signposts are lacking or else are vague and inaccurate. This is my excuse for opening this discussion. I shall endeavour to give a survey of the facts that seem to have emerged from research up to the present. I have exercised my discretion in picking and choosing, for not all that is printed is worthy of blind acceptance, but I have tried to be unbiased. To this I shall add some constructive ideas on our possible future policy. I expect, and hope, that both my conclusions and my suggestions will meet with fierce and searching criticism, which may winnow away the chaff of inaccuracy and false perception and leave behind perhaps a few grains of real truth.

The profession must approach this problem from two angles—that of clinical medicine and that of preventive medicine. They are two very different view-points and not

always harmonious. The clinician's main goal is the welfare of his individual patient, and if he wholeheartedly presses towards that goal he may not see eye to eye with the public health official, whose duty is rather to ignore the individual in his striving for the good health of the community. And since the problem of the child in the tuberculous household is both a clinical and a public health one, it must be approached from both angles; but any conclusions that are drawn, or any suggestions that are made, must satisfy the clinical conscience of the general practitioner and the preventive-medicine conscience of the public official.

I will bring the problem, as I see it, before you in a series of questions. I will endeavour to extract what I think are the true answers to those questions from the works of those who are researching on tuberculosis among children, and finally I will discuss the methods that have been or might be adopted as a result. In order to avoid the repetition of the rather clumsy phrase "the child in the tuberculous household," I shall refer to these throughout this paper as "contact" or "exposed" children.

FOUR QUESTIONS

The problem appears to me to be contained in four questions:

1. What is the relation of the incidence of infection, in time and intensity, among contact children to that among the general child population?
2. Does the contact child run greater risks of immediate sickness or death from tuberculosis than the average child?
3. Is the contact child exposed to greater risk of future sickness or death from tuberculosis than the average child?
4. Does the contact child constitute an immediate danger to the community?

I think that if we can find satisfactory answers to these questions we shall know the limits of the task before us.

What is the relation of the incidence of infection, in time and intensity, among contact children to that among the general child population?

In answering this question we find ourselves on fairly certain ground. For the purpose of our present discussion the end of the fifteenth year is taken as the limit of childhood. It is now universally agreed that, for practical purposes, the presence of skin sensitiveness to tuberculin is a proof of infection, and that a positive reaction means that somewhere in the body is a focus of tuberculosis, dormant and non-progressive perhaps, and living tubercle bacilli. It would seem possible that the lesions in question may be quite healed, since it has been shown by McPhedran and others that by annual inoculation living tubercle bacilli can be found in the tissues surrounding completely calcified lung foci and around fibrous apical scars in as many as 24 per cent. of the cases examined. In certain circumstances there may be no skin sensitiveness in infected subjects—for instance, in the later stages of acute and progressive tuberculosis, and during acute intercurrent diseases such as measles. But these exceptions are comparatively rare, and when dealing with large numbers of children, the skin reaction can be used as definite evidence of the presence or absence of infection.

The pioneer work in skin sensitiveness is associated with the name of von Pirquet and his scarification method. I think, however, I may safely state that his method has been superseded by the intracutaneous method of Mantoux by which 0.1 c.cm. of various dilutions of old tuberculin are injected into the skin. Von Pirquet's method is still used and upheld by some workers, but I will not weary you with the literature and figures contrasting the two methods, and simply refer you to Report No. 2 of the Brompton Hospital Research Department.

What is the incidence of infection among the average child population? This is an infinitely variable figure,

* Read in opening a discussion in the Section of Public Health (including Tuberculosis and Occupational Diseases) at the Annual Meeting of the British Medical Association, Eastbourne, 1931.

depending apparently on locality, general and tuberculosis mortality rates, race, and social environment. The highest known figures are those of Hamburger and Monti for Vienna, showing 94 per cent. infected at the age of fifteen, closely followed by Hetherington and others with 81 per cent. for Philadelphia. At the other end of the scale come Taylor's figures from Iowa with 31 per cent. infected at fifteen years of age. Heimbeck on 786 children between five and fifteen gives only 23 per cent., but he has used the Von Pirquet method. No. 2 Report of the Brompton Hospital Research Department gives the incidence of 1,000 children of the London working-class population of 59 per cent. infected at the age of fifteen. This may be taken as a fair average for that locality, but from the variations seen in the different parts of America it is probable that this figure might vary by 15 or 20 per cent. either way in different localities in England.

Contact children show a much higher incidence. As this is a definitely ascertained fact, I will quote only two examples. Opie for the Philadelphia district gives at the age of fifteen: Contact children 90 per cent. infected, non-contact children 64 per cent. The Brompton Research Department No. 2 Report gives: (1) average of all children, contact and non-contact, at the age of fifteen 59 per cent. infected; (2) non-contact children 52 per cent. infected; (3) contact children 82 per cent. infected. These figures speak for themselves, but analysis brings out another important fact. The figures show that by the age of fifteen the incidence is much higher among contact children, but closer investigation reveals that while in non-contact children the incidence increases steadily year by year from birth, in contact children the incidence reaches almost to its summit at a much earlier age. To quote Opie again, at the age of two and a half years among contact children 78 per cent. were infected, a figure very close to the 90 per cent. at fifteen. Among non-contact children 22 per cent. were infected, a figure far below the 64 per cent. at fifteen. The Brompton figures disclose the same, as this table shows:

AGE.	CONTACT.	NON-CONTACT.	ALL CASES.
	Per cent.	Per cent.	Per cent.
0-5	67	17	25
5-10	77	31	39
10-15	82	52	59

Therefore we may answer our first question by saying that although the incidence of infection is high among the general child population and varies in different localities, the incidence among contact children is much higher and the infection takes place much earlier, almost reaching its highest point during the first five years of life.

Does the contact child run greater risks of immediate sickness or death from tuberculosis than the average child?

Here again we can get a definite answer to our question. The death rate from tuberculosis, and therefore presumably the incidence of clinically recognizable tuberculosis, is definitely higher among contact children than among non-contact. Every investigator from every country agrees on this main point. But that is the only agreement they do reach. The actual figures produced are amazingly variable, and I do not propose to quote them. Some of the Continental figures indicate such a terrible mortality among contact children that one can only hope that they are incorrect. From our own country we have the evidence of Lissant Cox and others in Lancashire, with an experience of 1,486 children, and the Brompton Hospital Research Department Report No. 1 with an experience of 1,192 children of the London working-class population. Both these investigations show a definitely higher mortality from tuberculosis among contact children, but that as compared with the Continental figures the increase is small.

This statement needs qualification. It appears, though it is difficult to get accurate figures to prove it, that the risk of death is not equal through all the years of childhood but that it is much greater in infancy, that is to say, up to the age of three. Smellie, in the *Birmingham Medical Review*, quoted by Dixon, shows that the highest mortality in that district is between the ages of one and two years, and Opie and many others have stressed the danger to life of an infection in a child under one.

Those who have given thought to this aspect, notably, among others, Myers of Lymanhurst, are emphatic that the danger lies not in infection, but in continuous infection, when the effect of a non-lethal dose may be magnified by being received during the negative phase following a previous infection. Leroy Gardiner's work, published in the *American Review of Tuberculosis*, October, 1930, shows that after a first infection the negative phase probably lasts for at least fifteen days, and from four to six days after subsequent infections. I feel that the foundation of the doctrine of the danger of continuous exposure is probably sound. This would explain the higher mortality and greater risks of infancy, which is a time of more intimate contact with infected parents.

The cause of death is mainly, of course, generalized tuberculosis with meningitis. Smellie gives as the causes of death from tuberculosis in children: Cerebral tuberculosis 51 per cent., abdominal tuberculosis 33, pulmonary tuberculosis 14. The freedom of contact children from pulmonary tuberculosis is further suggested by Agassiz, who states that of 134 children under his care with pure pulmonary tuberculosis, in under 50 per cent. could evidence of direct contact be procured.

Although it has been stated that infection in an infant implies a bad prognosis, Myers, in support of the doctrine of the danger of continuous exposure, says that the prognosis is not bad; he presumably means if the infant is protected from future exposure after the first infection.

We may then sum up our answer to the second question by saying that the contact child is exposed to definitely greater risk of illness and death from tuberculosis than the average child, that the younger the child the greater the risk, and that it is suggested that the danger is due to repeated infections.

Is the contact child exposed to greater risk of future sickness or death from tuberculosis than the average child?

Here we get on to more debatable ground. The surest way to arrive at an answer would be by a "follow up" of a large number of contact children and noting the incidence of and mortality from tuberculosis among them as compared with the general population. This would be a stupendous task, as it would have to cover two generations of research workers and even then might be misleading, since the "followed up" children would certainly be more carefully supervised than the average child. As far as I know no such investigation has ever been attempted. We must try to get at it another way. Among 500 consecutive adult patients passing through the Brompton Hospital Sanatorium at Frimley a history of direct contact could only be obtained in 2.5 per cent. There is every chance of inaccuracy in such an enumeration, and even assuming that the figures are 100 per cent. inaccurate, they would still suggest that childhood contact, so far from being a menace as regards adult disease, may even exert some protective influence. Turning to pulmonary tuberculosis among adolescents, Dr. Lloyd has told me that among 208 consecutive cases of pulmonary tuberculosis in patients between the ages of fifteen and twenty he finds 31.5 per cent. who had direct childhood exposure, a much higher figure than that for the adult cases. But against this we have a statement by Dr. Lissant Cox that during a certain period not more than 1 per cent. of the cases

of pulmonary tuberculosis between the ages of fifteen and twenty-five in Lancashire had been sent to the dispensaries in childhood for previous examination. When we consider the careful way in which contact work is done in Lancashire, this can only mean that very few of these adolescent cases had direct childhood exposure.

The problem is a very difficult one. We must at all costs avoid suppositions and assertions, and confine ourselves to facts. For instance, Rathburn, quoted by Myers, states that 4 per cent. of those with the childhood type of pulmonary tuberculosis furnish 75 per cent. of the adult type of pulmonary tuberculosis of adolescents. I do not know on what he bases these exact figures, and certainly Dr. Lloyd's figures might seem to support him. But until we are certain, such statements are dangerous and must hinder progress.

Opie tells us that by twenty years of age 100 per cent. of the population where he has worked are infected, and the figure generally is certainly high. It is also certain that previous infection is necessary in any patient who suffers from the adult type of chronic pulmonary tuberculosis, but we have no evidence as yet that direct exposure in childhood in any way influences the incidence of pulmonary tuberculosis among adults.

Passing from the question of childhood exposure to the question of childhood infection, it is quite certain that a survived infection does carry with it a certain amount of future protection. This is beautifully demonstrated by some figures of Heimbeck. Of the nurses who were recruited to the tuberculosis hospital at Oslo between 1924 and 1928, 48 per cent. had been previously infected, as shown by a positive von Pirquet reaction, and 52 per cent. had a negative von Pirquet reaction (his figures might have been different if the Mantoux reaction had been used), and of 337 nurses in residence, of whom 152 were non-reactors, 53 developed clinical tuberculosis during their training, and of these no fewer than 51 came from the non-reactor class.

Therefore, the only answer we can give to our question is that childhood infection does exert a protective influence, but that there is no evidence to show that the extremely early infection, which is the feature of childhood exposure, exerts any influence on the incidence of adult disease.

Does the contact child constitute an immediate danger to the community?

Our knowledge of the exact vehicles for the transference of the tubercle bacillus from person to person is somewhat fragmentary. Calmette suggests that in any infected individual tubercle bacilli are excreted by the bile. It has also been demonstrated that there are non-acid-fast forms of the tubercle bacillus which still retain to the full their pathogenicity. The possibility of filter-passing forms has also been investigated. All these suggestions bear importantly on the question of infectivity, but as long as they remain in the realm of hypothesis we cannot use them to answer our further question. We must say that, as far as our own present knowledge goes, the contact child not suffering from clinical tuberculosis is not a public danger. Before we can say that they are carriers and spreaders of infection we must wait for future research.

To sum up the position:

1. Tuberculosis infection is very common among children.
2. The incidence varies widely in different localities.
3. It is much higher among contact children.
4. It takes place earlier in life among contact children.
5. The mortality from tuberculosis is higher among contact children.

6. Infection is more dangerous to infants.

7. It is more dangerous if there is continuous exposure.

8. Childhood infection exerts a protective action as regards adult tuberculosis.

9. Infantile infection exerts no recognized influence on the incidence of adult tuberculosis.

10. The influence of infantile infection on the incidence of adolescent tuberculosis needs further investigation.

11. Contact children, as far as we know, do not constitute a public danger as spreaders of infection.

CONSTRUCTIVE SUGGESTIONS

Now what constructive suggestions can be made on the basis of these postulates? I have never myself regarded the problem of the contact child to be so all-important from the preventive medicine point of view. Sir Robert Philip ascribes the decreasing mortality from tuberculosis at the present day to our active anti-tuberculosis legislation and activity, but tells us that we are concentrating too much on the pronounced lesions, notably pulmonary tuberculosis. But the decline in infant mortality during the past twenty years has been immeasurably greater than the fall in the general mortality, or than the fall in the mortality of any other age period; it has been more than twice as great. If the fall in the general death rate may be ascribed to our efforts, may not the greater fall in the infantile death rate be ascribed to them too? And may it not be that by tackling the problem of adult pulmonary tuberculosis, and by continuing to do so vigorously, we are making our strongest attack on infantile tuberculosis and tuberculosis as a whole?

But, however much we concentrate on adult tuberculosis, the problem of the child in the tuberculous household will still confront the general practitioner and the public health officer.

The general practitioner must view this mainly as a clinical problem; his duty is towards the child and its parents rather than to the community at large. Is he to examine all the contact children in his practice by the Mantoux reaction for evidence of infection and by the x ray for evidence of lesion? No, he should not have to deal with these personally; but these investigations should be made for him, and he should have a knowledge of their results. His duty is to watch, and to turn his attention to the proper hygiene of the home. The importance of this will be realized when we learn from Sir P. Varrier-Jones that there is a complete absence of clinical tuberculosis among 133 children born at the Papworth settlement. He must realize the danger not of exposure but of continuous exposure. That is to say, he must use every effort to sterilize the open case which is the source of contact, and to dilute the infection either by treatment or by segregation, partial or complete. Contact infants must be his especial care in this direction. He must teach the parent and the adult how by attention to a few simple hygienic rules they can minimize the danger to the children almost to the vanishing point. It is not his duty, and would be a waste of his time and injurious to his practice, if he were to say to the tuberculous mother, as did one practitioner of my acquaintance, "You must never see your child again." When the contact children of his practice reach adolescence, then is the time he must be most wise and wary. That is the time when the parent and child need guidance in choice of work, life, and environment. That is the time when the x ray will be particularly useful, for McPhedran has said (and he is a man to be listened to) that apical infiltration in childhood and adolescence, although apparently latent, always goes on to definite clinical pulmonary tuberculosis if untreated.

The public health officer will stand behind the practitioner to supply him with such facilities as will help him in dealing with this problem, or to step into the breach with these facilities in the interest of the patient if the practitioner or the parent is neglectful of his duties. I think that he will be wiser if he, too, views the problem from the clinical standpoint. For the answers to our questions fail to show that the contact child is an important public health problem. It will be the public health officer who will draw the attention of the general practitioner to open cases of tuberculosis which are the source of contact to the children in his practice. I would like to see a tuberculin reaction done on every child when it reaches a certain age. It certainly should be done on every contact child, and every positive contact reactor should have the thorax examined radiologically, and the information thus obtained should be filed and recorded for future information and research and should be passed on to the general practitioner. This is essentially the work of the specially trained public health officer. Further, he should be ready to re-examine such children whenever the practitioner thinks it is necessary, and be ready at all times with his special knowledge of the disease and with the facilities at his disposal to give any advice or help that may be required.

These suggestions on the attitude and the duty of the general practitioner and the public health officer toward the contact child may be well enough, but save for the plea for the more extensive use of tuberculin as a diagnostic agent and the *x* ray, they do not contain anything new. What can the results of present-day research, as we have seen them, suggest in the way of improvement? We provide institutional accommodation for the definite case needing treatment. But what exactly is the definite case needing treatment? The boundary between infection and clinical disease is ill-defined. Do we go near enough to that boundary in choosing our cases that need treatment? What types of lesions are likely to develop into clinical disease? The adolescent with open and obvious pulmonary tuberculosis is a pretty hopeless problem. If only we knew more about the danger signals, could we not avoid this catastrophe more often? These are, I think, some of the great gaps in our knowledge; and it is in this direction to-day that we need guidance, and in which future research should proceed.

If the answers to these questions are forthcoming, perhaps more cases will be found to have definite clinical disease and to be in need of treatment, institutional or otherwise. But how shall we deal with those who only need preventive treatment? The so-called preventorium is largely used in Canada and the United States, and perhaps we might make more use of it in England. But if we do it must be properly used. I quote from Dr. J. B. Hawes junior, writing in the *American Review of Tuberculosis* in July, 1929, about the Prendergast Preventorium; he says:

"As I study the results of 110 children discharged from our Preventorium I am more convinced than ever that the ultimate success of preventorium treatment depends upon thorough, aggressive and prolonged follow-up work. No child should ever be allowed to return to a home where it will again be exposed to infection. This should be an absolute rule."

This statement appears to me, if it is a true conclusion, to show that a preventorium is absolutely useless, at any rate as it is conducted in that district. The expense and trouble of such an institution might be saved and, to use Dr. Hawes's words, a *thorough, aggressive and prolonged* Grancher system might be substituted instead, or the rigorous enforcement of some such harsh law as they have in Chicago forbidding children to live in the same

house as an open case of tuberculosis. No, it does not seem that a limited course of institutional treatment on sanatorium lines can help the problem in any way. A preventorium that I saw in use in Toronto seems to meet the case better. There, contact children are placed, provided they show no evidence of active clinical tuberculosis, and kept and trained and taught until the tuberculosis authorities are satisfied that their home conditions have been rendered as satisfactory as possible. The average stay is three to four months. That seems a reasonable method of spending public time and money for the benefit of the contact child. In general, short courses of enforced hygienic life, and harsh interference with family liberty and happiness, do not appeal to me.

B.C.G. Immunization

Are we going to get help from immunizing processes? Is Calmette's B.C.G. the weapon of the future? Now we must deal very cautiously with B.C.G. It seems to arouse the controversial spirit. There are two questions to be answered. 1. Is it harmless? 2. Does it protect?

The answer to the first question is not certain yet, but we can say that for practical administration, if used according to the technique of Calmette and Guérin, it is harmless. The adverse reports and accusations of its toxic possibilities have come mainly from pure bacteriologists and laboratory investigations, and as far as I can see have no very great practical bearing. Does it protect? In cattle apparently yes, to a certain extent. I must confess that the French figures on the vaccination of children leave me confused and unimpressed, but I should like to quote Heimbeck again, this time on the protective power of B.C.G. Still dealing with the nurses coming to tuberculosis work in Oslo, he found:

That of 322 non-reactors to tuberculin who were inoculated with B.C.G., 294 became reactors.

In 1924 there were 8 cases of tuberculosis among non-reactors.

In 1925 do. 25 do. do. do.

In 1926 do. 14 do. do. do.

In 1927-28 among 44 non-reactors who were vaccinated there were no cases of tuberculosis, and among 12 non-reactors who refused inoculation there were five cases.

He summarizes by saying that previous to 1927 the morbidity among non-reactors was 16 per cent., and in 1927 among vaccinated non-reactors 0 per cent. These are very striking figures. And it is to be noted that a very large percentage of the non-reactors became reactors after vaccination.

These figures do suggest that B.C.G. vaccination may be a useful weapon to tide over the dangerous period of infantile contact and exposure, in conjunction with other methods for preventing continuous exposure to massive infection. These other methods are necessary, for a first infection with ordinary tubercle bacilli or with B.C.G. will confer no immunity that can overcome a massive infection or continuous intense exposure.

Whatever protection is afforded by B.C.G. is admittedly short-lived, and continuous vaccination throughout childhood of all contact children is an impossibility, but its use on infants might convert the unprotected non-reactor into the partially protected reactor. Myers has stated that in children a high degree of hypersensitiveness to tuberculin is a dangerous sign. This suggests, for those who cannot bring themselves to support the use of B.C.G., the use of the admittedly harmless tuberculin. It would be quite possible for those contacts who showed what was agreed to be a dangerous degree of hypersensitiveness to be desensitized by a course of tuberculin injections.

I repeat that I do not think the problem is so very grave a one as it sounds, and it seems to me that the future safety of our children depends on the improvement in

personal, familial, and community hygiene; and what vistas of possibility are opened up by Professor Mellanby's lecture on the prevention of disease by the correct feeding of children. It is obvious that we must not try to escape infection for our children, for, dangerous though infection is, it does bring with it protection, and we must avoid the state of affairs that Heimbeck deplores in his own country in these words:

"Thanks to tuberculosis work, infection in the home is not so common and is postponed until the individual goes out to work. Thus the endeavour to avoid tuberculosis infection in childhood has resulted in the transference of a high morbidity and mortality to just those years when the individual should begin to work for society in return for what society has done for him in the matter of education in his earlier years."

Really, it is Nature's problem. She will find her own solution, and I think she is solving it, as Dudley Sheldon suggests when he says:

"Morbidity equals infection pressure versus herd immunity. Herd immunity increases with the herd's past experience of the bacterial cause of infectious disease, and although infection pressure must be greater with the increase of urbanization, herd immunity outstrips it."

THE BROKEN NOSE*

BY

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(With Special Plate)

A broken nose is common enough and regarded often, except perhaps by the victim, as matter for mirth rather than for sympathy. Fortunately, even in these motoring days, the majority of our cases are of a mild type: when there is no subsequent external deformity, any distortion of the nasal septum can be readily dealt with by sub-mucous resection. Omitting those in which there was no manifest displacement of the nasal bones, the etiology in twenty-seven cases treated personally during the four months March to June, 1931, was as follows:

	Mild	Severe
Football	3	—
Cricket	4	—
Boxing, etc.	1	1
Motoring	2	4
Cycling	1	1*
Falls, etc.	3	1
No record	6	—
	20	7

* Collision with motor lorry

But the deformity resulting even in the milder cases may be very annoying—to a girl possibly really distressing. If it is to be avoided it is important that treatment should be undertaken *early*, since the processes of repair lead to rapid absorption of bone edges, and callus formation may produce an unsightly bossing along the margin of the frontal process of the maxilla; so that a good result is often not obtained by late treatment, even though the bones be re-fractured and set. Still more important is it that treatment should be intelligent—that is, guided by a proper understanding of the exact nature of the deformity, and the best method of reduction. Though it is often easy to see the line of fracture in an ordinary skiagram, the actual displacement in a mild case is not well shown unless resort is had to a stereogram. Gross damage is, of course, sufficiently obvious in any good film (Figs. 1 and 2 on Plate).

* A Bristol University Post-Graduate Lecture.

In a typical mild case the accident is followed immediately by epistaxis, and very soon by nasal swelling, internal and external, probably with a black eye in addition; it is impossible in these circumstances to estimate damage. Local treatment is confined to suture of lacerations, etc. In four to eight days the swelling has gone down, nasal obstruction is slight or absent, and one can make out the characteristic deformity (Fig. 3 on Plate). The bony nose is bent definitely over to one side, the septum and tip being little affected. If now one attempts simply to push the nose back into place, in all but the most trivial cases this is found unexpectedly troublesome, and a symmetrical appearance is not achieved (Fig. 11). Also, the deformity tends obstinately to recur; internal splints effect nothing, while external splints or padding are often impossible to retain in place. If the true mechanism of the fracture-dislocation is appreciated, reduction is perfectly simple, and one can almost guarantee that there will be no subsequent deformity.

The actual displacement of bone presents three features (Figs. 9 and 10).

1. On the side from which the blow fell the lower two-thirds of the nasal bone, and often the anterior sharp margin of the frontal process of the maxilla, are displaced inward toward the mid-line, mainly by rotation round the posterior margin of the fragment as a hinge, with very little backward displacement.

2. On the opposite side a corresponding piece of bone, or rather less, is displaced in the same direction—that is, outward—again hinging on its posterior margin.

3. In the middle the first fragment has been driven in beneath the second. It is lack of appreciation of this imbrication-impaction that is the source of difficulty and failure in reduction. Figs. 9 to 13 are diagrammatic sections at right angles to the plane of the nasal profile, to explain the common types of displacement and the steps in reduction.

TREATMENT

A general anaesthetic is necessary, and oxygen-ether will be found convenient. Bleeding is usually slight; if desired, the nose may be lightly sprayed just before induction with 5 per cent. cocaine solution and packed anteriorly with gauze wrung out of 1 in 10,000 adrenaline. When anaesthesia is complete a post-nasal sponge can be introduced. The only instrument needed is a strong forceps, the blades 2 to 2½ inches long and slightly bowed so that only the tips touch. Jansen's longest bone forceps may be used; the common septum forceps is too thick for most noses. Before use the tips are well padded by winding on three or four turns of half-inch strapping.

Operative Procedure

A.—Into the nostril of the side *toward which* the nose is bent pass one blade of the forceps, pushing it up until the other blade outside the nose is just over the nasal bone. Grasp the bone firmly and twist it yet further outward, still round the posterior margin as an axis (Fig. 12). (This step is not always essential, but greatly facilitates disimpaction.)

B.—In exactly the same manner grasp the other or in-driven bone, and rotate it also well outward to a position corresponding with the first (Fig. 13). Sometimes a slight lifting movement is required in addition.

C.—A finger placed externally can now feel that both bones are freely movable, and that there is a definite gap between them anteriorly. With the finger mould the two bones symmetrically together, like the leaves of a double drawbridge, so that they lock in the mid-line. If necessary, the forceps may be used with one blade up each