real cause of the trouble. It is curious that the majority of inquirers, who discredit the reputed ill effects of consanguineous mating, consider any suggestion of it similarly "begging the question," and this view of the matter is either ignored or derided by recent writers on the subject. The idea is, for instance, only touched upon at one point by Myerson,⁶ who writes:

"I scoff at the implication that because the paternal great-grandfather and great-grandmother were first cousins that we have any clue whatever to the gross defect in their great-grandchildren, their father and mother being superior people."

He would probably similarly scoff at the suggestion that the continuous mating of cousins—irrespective of any imaginary common "taint"—could tend to produce dementia praecox, but such a result is surely possible.

Some writers accept as ancestral "taints" such conditions as, for instance, heart disease, blindness, and neuralgia! These inquirers do not apparently realize that we are all imperfect, and that the fact of such imperfection in an ancestor is not, after all, a satisfactory proof of the likelihood of mental disease occurring in a descendant.

E. Rudin⁷ considers that dementia praecox is transmitted according to Mendel's law, and is increased by inbreeding. Myerson,⁶ after able analysis of many family groups, agrees with Rosanoff,⁸ Jolly,⁹ Luther,¹⁰ and Krueger,¹¹ and states that "dementia praecox breeds true . . . and in an ancestor is usually followed by dementia praecox in the direct insane descendant. Moral imbecility, feeble-mindedness, and epilepsy are also found in the descendants and frequently in combination with dementia praecox." In other words, he considers dementia praecox a Mendelian recessive character. The writers I have mentioned find, further, that insane descendants of persons who have suffered from the involution psychoses are cases of dementia praecox, as are also often the insane descendants of manic-depressives. As Myerson⁶ puts it, "All roads seem to lead to dementia praecox and from thence to imbecility. As the disease gets worse from generation to generation it ends in dementia praecox." Dementia praecox in an ancestor, on the contrary, does not appear to be a precursor of manic-depressive in the descendant, but as deterioration becomes more marked dementia praecox appears, and I can imagine no condition showing such obvious all-round degeneration as this syndrome presents.

If many of the original refugees had been schizophrenics, it is very doubtful if a Parsee race would now exist in India. But 100,000 do exist (among some 320,000,000 Indians of all races and classes), and include a large proportion of very able persons. At the same time most of those who become insane suffer from dementia praecox, and the custom of the marriage of cousins has persisted since the eighth century in this community, which is not a large one. Judging by the results obtained by Myerson and others, it is therefore probable that any original taint which, by its transmission through inbred generations to present-day Parsees, might breed in them dementia praecox, was itself a very much less serious form of mental disorder—if indeed it could be classed as such at all.

Our present-day differentiation of mental deficiency from insanity is surely merely one of convenience, and few will, I think, argue the impossibility of the consideration of schizophrenia as being a variety of the former rather than of the latter group of disorders. In most cases the degenerative tendency appears to be inborn in the individual.

As I have previously written, the disorder has a predisposing and an exciting cause. In the case of the Parsees the former is contained, presumably, in the heredity, but it is, I think, a question whether the heredity acts as a cause solely by means of an inherited taint or whether consanguineous mating in some way itself lessens resistance to the stresses of life, and helps to produce the disorders of metabolism that are possibly the most usual exciting causes of the breakdown. As White¹² puts it:

"Every individual born into the world has, if it could be determined, a definite potentiality for development. The force or the impetus which starts it on its path is sufficient to carry it a certain definite distance. In the subject of this disease the original impetus has been weak, and when its force is spent [mental] development stops and the retrograde process is hastened or initiated by some special physical or mental stress occurring at the critical point of puberty and adolescent evolution."

What is, however, the cause of the failure to carry on? Tredgold¹³ comes to the conclusion that feeble-mindedness results from a vitiated germinal vitality which prevents normal development, and most authorities are, as Myerson⁶ writes, "firmly convinced that there is a familial transference of certain types of feeble-mindedness, and there is very distinctly appearing the belief that these familial cases originate in injury to the germ plasm." One may perhaps speculate on the kind of injury he adumbrates. It is possible that "similar" germ plasms may be in some way detrimental to each other.

Most authorities insist on the view that the marriage of cousins conveys no danger to the offspring, if the common stock is sound. There is, however, no reason to suppose that the original refugees were in any way more unsound than any similar group, and, as no stock appears to be absolutely sound, the contention that consanguineous mating should ever be approved is very doubtful, as it must tend to intensify in the offspring any taint common to both parents. Nowadays at all events, the common Parsee stock is seriously tainted with dementia praecox, and the propriety of the abandonment of the custom of the mating of cousins should be definitely considered by them. Perhaps research into the results of such mating may be possible in some other field, but I am certain that little further result can come of inquiry in this one, unless it is carried out by a Parsee of an unusually altruistic type. The subject is one of immense importance sociologically and eugenically, as dementia praecox is the form of mental disorder which, owing to its relative incurability, keeps our mental hospitals full.

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AUTO-TRANSFUSION.

WITH THE ACCOUNT OF A CASE.

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ALTHOUGH auto-transfusion may often be the best and most readily available means of saving life in a surgical emergency it has not received the notice which it deserves, and the object of this communication is to draw attention to so useful a measure. Our interest in it was aroused by the following case.

A boy, aged 12, fell from the balcony of his home to the pavement beneath. He was perfectly conscious, although he had a very local fissured fracture of the skull, subsequently revealed by x-ray examination, and both bones of each forearm were broken. His pulse rate was slow. The abdomen was soft and not tender, and the patient assured us that he had received no injury in that situation. Under ether anaesthesia the fractures of his forearms were reduced. A few hours later signs of severe intra-abdominal haemorrhage definitely showed themselves. He rapidly became blanched and pulseless, and was in a desperate condition when brought to the operating theatre. The haemorrhage came on insidiously, because its early signs were doubtful and obscured by the other injuries and the anaesthesia.

Always ready in our operating theatre, even to the smallest detail, is what we call our "transfusion tray." Realizing the possibility and value of auto-transfusion in such a profound bleeding, this ready-to-use tray was requisitioned, and proved the great value of such a precaution, because of the short time at our disposal. The abdomen was opened by Mr. Devine, and found to be full of fluid blood containing a few clots; this was quickly ladled out and dropped into citrate solution. Meantime a vein had been opened by Dr. Kinsella, and reinfusion of the citrated blood was begun even while the blood was being removed from the abdomen. The source of bleeding was found to be a tear near the pedicle of the spleen. The abdominal wall showed no sign of trauma, the injury having been inflicted apparently in some indirect way.

The bleeding appeared to stop as the transfusion was given, and gauze packing was gently introduced over the tear. Thanks to the large quantity of blood which had been infused, a dramatic change now took place. The patient became quite warm, his colour improved, and the pulse became easily palpable. The packing was watched for a time, with a degree of equanimity impossible in the early stage of the operation; the abdomen was closed and the patient returned to bed. No reaction followed, and convalescence was rapid and uneventful.

This gratifying result suggests the advisability of using reinfusion in the more severe cases of acute intraperitoneal bleeding, in which the patient urgently needs an addition to the circulating fluids. It is not uncommon to see such patients given various solutions by different routes, while the most suitable fluid of all, their own blood, has been thrown down the sink. Experience has shown that blood can be safely reinfused, even after lying in the abdomen for a considerable time. The question of changes taking place in the blood does not arise in the acute haemorrhage for which this measure is advocated, for the blood is comparatively fresh in these cases. The hurried submammary saline given in the operating theatre, or perhaps in the ward, may sometimes lead to undesirable infection, but we have not seen an infective complication following an intravenous injection. Probably the reason is that blood is not only an unsuitable medium for the growth of bacteria, but has marked bactericidal effect, as shown by Almroth Wright, Colebrook, and Storer.¹ It is known that germs may enter the blood stream, and their growth be kept in check until they are finally excreted by the kidney or by the liver. The slight infection possibly present in acute ruptured ectopic gestations is not necessarily, therefore, a contraindication to reinfusion.

A German gynaecologist, Thies, is given the credit of introducing auto-transfusion in 1914.² Burch published a survey of the literature in 1923.³ Up to this time 164 cases had been reported, 160 of these in Germany, made up chiefly of acute ruptured ectopic gestations and severe injuries and wounds with haemoperitoneum and haemothorax. In these cases there were 14 post-operation deaths, only one of which could possibly have been caused by an infusion. The method was proved to be valuable and safe.

Sir William Taylor of Dublin suggested a further use for reinfusion in splenectomy.⁴ This operation may be very difficult, and attended by much blood loss and shock, especially when the spleen is large and the patient is already suffering from blood dyscrasia. As soon as the spleen is removed it is held by an assistant over a vessel containing citrate solution. The clamp is removed from the pedicle, and the blood is allowed to drip into the vessel, the flow being aided by squeezing the organ. This citrated blood is then reinfused.

Loyal E. Davis and Harvey Cushing found reinfusion a most valuable and life-saving device in the major cranial operations.⁵ The blood is removed from the field of operation by suction rather than by swabbing; citrate solution is sucked up from a basin, swabs and towels are wrung out into the solution, and the mixture is reinjected. The authors show impressive graphs of very severe surgical shock in which immediate and marked improvement followed this reinfusion.

Scant attention has been paid to this method in British literature; in discussions on blood transfusion it is summarily judged and condemned without trial. Thus, in a recent discussion its only notice was a brief reference by Sir Humphry Rolleston to Blain and Brines, who believe that reinfusion is inadvisable.⁶ But Blain and Brines record no more than their beliefs, give no reasons, and have apparently no experience of the method.⁶

Technique.

In giving intravenous injections of large volumes of fluid every effort should be made to prevent those reactions which the patient so often suffers after this treatment. In auto-infusion we are relieved, of course, from every anxiety about the compatibility of the donor.

Probably the next most important point, in giving any intravenous injection, whether of blood or of saline, or of glucose, is to exclude not only living but also dead organisms. The importance of keeping out dead bacteria is at once suggested by experience with intravenous vaccinations—for example, in the treatment of typhoid

fever and of rheumatoid arthritis. The reactions following such treatment and the usual post-transfusion reactions are similar in every way. They appear a few minutes to half an hour after the injection; the patient feels cold and has a rigor, which may be very severe. In practice, therefore, it is not enough that the water used in making the solution should be sterile; it should be freshly distilled before sterilization. Whenever possible it is our practice to have the water distilled on the same day that the injection is given. For emergencies a supply of water, first distilled, then sterilized and sealed, is kept on hand. This should keep indefinitely without bacterial contamination, but, as a routine, it is boiled before use. In two and a half years we have experienced only three marked rigors after infusion of blood or glucose. In one of these glucose dissolved in ordinary tap water had been given in an emergency, and in another blood transfusion had been performed with sodium citrate dissolved in the chemist's stock distilled water. The importance of using freshly distilled water in blood transfusion has not been sufficiently emphasized in the literature; this probably accounts for the unduly high proportion of reactions which follow the use of citrated blood. Even Lewisohn, the great protagonist for this method, reports 23 per cent. of reactions.⁷

Burch states that the use of citrate solution in auto-transfusion is not necessary, for the blood has been given mixed with saline, or defibrinated, or even unmodified. But the citrate method is the most certain, for it eliminates the possibility of clotting, which might cause serious delay or even failure in desperate cases. Moreover, it enables one to give the infusion slowly, and this point is most important, not only to avoid overloading the heart, but also to help to prevent undesirable reactions. French observers (Cruchet and Ragot) declare that even if agglutination and haemolysis take place, no ill effects need follow if the injection is given slowly.⁵ An ounce a minute is the maximum speed permissible when using compatible blood or a suitable solution.

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Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

TRAUMATIC ASPHYXIA.

TRAUMATIC asphyxia is rare, and some of the standard textbooks on surgery and medical jurisprudence do not even mention it. The following details of a case may therefore be worthy of record.

In 1926 a tall, powerfully built young man, aged 28, presented himself at my consulting room. His face was deeply cyanosed and the subconjunctival tissue of both eyes was suffused with bright red blood, giving him a most startling appearance, although he did not appear to feel ill and had walked half a mile to see me. He stated that except for a headache he had been in good health when he went to bed two nights previously; he awoke the following morning in his present condition. He and his family absolutely denied that he was subject to fits, and they could give no clue as to the cause of the seizure. He mentioned that he had suffered from a similar though less severe attack two years previously. He had consulted me seven months before for a small subconjunctival ecchymosis of one eye for which he could ascribe no cause.

On examination it was found that, above a line running round the neck from the level of the upper margin of the cricoid in front to the fifth cervical spine behind, the skin of the head and neck had a deep cyanotic tint, with a few small points of ecchymosis in the scalp behind the ears and over the cranium. The mucosa of the palate, tongue, and pharynx was also of a purplish hue and showed several small extravasations of blood. Below the line of demarcation the skin was perfectly normal. There was no mark of any constricting band.

In the absence of information as to the mode of onset I could not explain the condition until, a few days later, I learned from