A Paper

ON

THE DISTRIBUTION AND NATURE OF
FOOD POISONING.*

BY

JOHN CRUICKSHANK, M.D.,

PROFESSOR OF BACTERIOLOGY, MARISCHAL COLLEGE, ABERDEEN.

THE subject of bacterial food poisoning presents many features of interest to bacteriologists and practitioners. The sporadic nature of the outbreaks, the dramatic onset of symptoms, and the frequent difficulty of tracing the cause make an unusual appeal to the medical mind. As an additional interest the outbreaks are described in glaring outlines in the daily press, through the medium of which the knowledge of the outbreak is frequently first disseminated. The character of the outbreaks, particularly in their unexpectedness both in time and locality, presents difficulty to the examining bacteriologists, and prevents that continuity of research which is so essential in the investigation of obscure disease. Cases are frequently scattered over a large area, and correlation of the results is correspondingly difficult. Except for the work of a few men, systematic investigations of the etiology of these cases has not been carried out, and as a result the subject presents many unsettled problems.

In opening this discussion I cannot attempt a complete review of the subject, and must content myself with the examination of certain aspects only. To my knowledge there is no new work of special importance to communicate, and it is to be hoped that in the discussion which follows some new information may be given which will throw light on the problems.

It is impossible to approach the subject without an appreciation of the work which has been carried out by Dr. Savage, whose painstaking collection and analysis of outbreaks in this country has added to our knowledge, and whose views, particularly with regard to etiology, will probably serve as the nucleus of this discussion. His interest in the subject and his researches have served to focus the attention of workers in this and other countries upon the whole field of bacterial food poisoning, and constant reference to his work is unavoidable in the course of this paper.

The characteristic features of bacterial food poisoning are the sudden onset of sickness, vomiting, severe diarrhoea, abdominal pain, and pyrexia. The interval between the taking of the infected food and the onset of symptoms is short, varying from two to thirty-six hours. In the majority of cases the severe symptoms persist only for a short time, and within twenty-four hours the patient suffers only from exhaustion. Pyrexia is of short duration, may be absent, and subsides with the severity of the alimentary condition. It is of interest to note that even when the diarrhoea is very severe blood and mucus are rarely found in the stools—a feature of interest in distinguishing these cases from severe dysentery. The extremely short incubation period in some cases has suggested that the symptoms result from the presence of preformed toxins, as it is not in accord with general experience to believe that the viable organisms in the food which is ingested can themselves proliferate within the alimentary canal in sufficient numbers to produce severe reactions in such a short time. In certain outbreaks there is clear evidence that the suspected food has been subjected to such exposure to heat as would ensure the death of all vegetative forms of bacteria. As the signs and symptoms which have occurred in these cases are very similar to those in which live organisms have been demonstrated it has been suggested that heat-resistant toxins are present. This aspect of the subject has recently attracted much attention, and has been examined by experimental work on animals.

Since 1888, when Gärtner described the organism which bears his name as the causative agent in an outbreak of food poisoning, the attention of observers has been con-

centrated upon the demonstration of this or related organisms in cases of food poisoning associated with the classical symptoms of enteritis. With the development of bacteriological methods and the institution of more intensive examination of such cases, a group of organisms possessing similar morphological and similar, or closely related, biochemical characters has been described of which some members have been concerned in the causation of food poisoning in man or with severe enteritis in animals. The close relationship of these to the paratyphoid organisms has been repeatedly emphasized, and highly technical procedures have been developed in the attempt to differentiate the various members of the group. The general term "paratyphoid-enteritidis" has been applied to this group, although certain workers prefer to use the term "salmonella group," after the hog-cholera organism described by Salmon and Smith. The organism most frequently found is not Gärtner's bacillus, but *B. aertrycke*, an organism very closely allied to *B. paratyphosus* B, and only distinguishable from it by the test of absorption of agglutinins. Since the relationship of this organism to food poisoning has been established its presence in animals closely associated with man has been demonstrated, and the discussion of human food poisoning is closely bound up with the studies of the occurrence of this and related organisms in the intestines of rats, mice, pigs, sheep, and cows.

It may be said that the list of food-poisoning organisms is constantly being extended, largely because of the extension of bacteriological methods of examination to the question of the antigenic structure of bacteria. These methods have proved fruitful in the separation of types hitherto indistinguishable, but have in many ways made the problem of food poisoning more difficult. The work of Sir Frederick Andrewes on the subject of specific and group phases as applied to agglutination reactions, and the great development of cross-agglutination and absorption tests, has not resulted in simplification of the subject, and has made it very difficult for the routine bacteriologist to identify clearly the strain of organism which he may have isolated from a particular outbreak.

Food poisoning has occurred in many countries, and in the general study of these outbreaks it is necessary to take into account the habits of the people as regards food and the general standard of sanitation of the country, particularly with regard to the slaughter of animals and the preparation of food for the table. An important feature of most outbreaks is the apparent soundness of the suspected article, which neither in colour, appearance, nor taste may have seemed to be different from the sound article. This factor has operated adversely in the subsequent examination of the cause of many outbreaks, as it is difficult to secure samples of the suspected food, which has all been consumed. In these cases, unless an organism of the salmonella group can be isolated from the patient's vomit or stools, the cause of the outbreak may remain obscure, although indirect evidence of suggestive character may be obtained by methods such as the examination of the blood of convalescents for the presence of agglutinins for the salmonella group. A very puzzling feature of some outbreaks is the small number of individuals affected, although the infected material—for example, the carcass of an animal—has been widely distributed through a community. In the attempt to explain these outbreaks it has been necessary to suppose either that individual factors of susceptibility play an important part or that the infecting organisms have gained entrance to certain portions of meat through the medium of mice or rats. According to Savage, human carriers, who are so important in the spread of enteric diseases, are not to be regarded as playing an important part in infection, but the possibility cannot be excluded.

A wide variety of foodstuffs has been found to be connected with the outbreaks: tinned meat, salmon, sardines, milk, meat, sausages, pork, mutton, and made-up foods of various kinds. The possibility of infection from eggs has been raised in connexion with a recent case, and Doyle has shown that severe diarrhoea in chickens and ducks may result from infections with *B. aertrycke*. Of these articles the only one which seems to offer conditions suitable for growth of salmonella organisms is milk, and there is

*Read in opening a discussion in the Section of Pathology and Bacteriology at the Annual Meeting of the British Medical Association, Manchester, 1929.

evidence that where milk is the vehicle the outbreaks are widespread and severe in character. In the case of beef, mutton, and pork, suspicion frequently falls upon the animal from which the meat has come, which during life may have been the subject of salmonella infection. The way in which tinned foods such as salmon and sardines become infected is not clear. I am not aware that fish suffer from salmonella infections, and it is necessary to suppose that in the preparation of these foodstuffs, previous to "processing," infection has resulted in the case of individual tins from some unknown source, and that considerable growth of the organisms has occurred with the development of "toxins" which are resistant to subsequent heating.

I have much difficulty in following the chain of reasoning which has been presented as a probable explanation in many outbreaks. It is easy to understand infection of the human subject with living organisms, even though few in numbers, but when these are apparently absent and the outbreak is attributed to heat-resistant poisons an interval for proliferation of the organism and production of sufficient toxin must be postulated. In many of the outbreaks there is no evidence that these conditions are present. It is inconceivable that a few dead organisms in a sample of food can be held responsible for the pronounced symptoms of food poisoning. Experimental work with regard to the ability of salmonellas to grow in various types of foodstuffs at the temperatures at which these are kept would throw light on this aspect of the subject. When the interval between the taking of the food and the onset of symptoms is very short the presence of preformed toxins is suggested as the cause, whereas in the cases with longer incubation period the symptoms are referable to the development in the alimentary canal of viable organisms. It is supposed that both preformed toxin and live organisms may, under certain conditions, be present, and that the symptoms produced by toxins alone may be as severe as those produced by the development of living organisms.

With regard to infection from the meat of animals which have been the subject of salmonella infection during life there is no evidence that their muscular tissues are so heavily infected that after cooking the few organisms present in a small amount of meat could evoke the severe manifestations which occur in food poisoning. It is the opinion of various veterinary workers that the most likely source of infection at the slaughterhouse is from the soiling of meat with intestinal contents. If this is correct, the degree of infection—that is to say the numbers of organisms transferred from an infected animal to other meat—must be extremely small. If we accept the view of Savage that heat-stable poisons from the salmonellas are the cause of these outbreaks, we must suppose that the organisms have developed in large numbers between the death of the animal and exposure to cooking.

The relatively infrequent finding of Gärtner and *B. suipestifer* types is an important feature in the study of these outbreaks, since these types of salmonella appear to be the most frequent exciting agents of disease in cows and pigs. The occurrence of *B. aertrycke* types in the diseases of food animals is extremely uncommon, yet the *B. aertrycke* type is the most common finding in human outbreaks. In the 100 outbreaks recorded by Savage and Bruce White the presence of living *B. aertrycke* was demonstrated in 16 outbreaks, *B. suipestifer* (types Reading and Derby) in 2, and Gärtner only in 1. Many of the outbreaks were investigated in very incomplete fashion, but the absence of living organisms in such a large number of cases would suggest that other causes than salmonella infection should be kept in mind. Unless we suppose that infection of food animals with *B. aertrycke* is more common than has hitherto been shown to be the case infection of the food must have resulted either from human carriers or from contact with animals of the vermin class, particularly mice.

The view strongly advocated by Savage that salmonella infections account for all the cases of food poisoning, with the exception of the few in which chemical poisoning can be shown to be the cause, has led to the concentration of investigation upon the discovery of this group of organisms, with the result that other possible bacterial causes may be missed. In most of the recorded cases

there is inadequate data regarding the bacteria present in the food, and in the examination of the stools the presence or absence of salmonella group organisms only has been considered. In the case of dysentery, for example, it has been necessary in recent years to admit to the group of pathogenic organisms the Sonne type of dysentery bacillus, whereas formerly only Flexner and Shiga types were considered in relation to the disease; and it cannot be said that the list of pathogenic organisms in acute disturbances of the alimentary canal is closed when so many cases of diarrhoea, particularly in children, give negative results by the accepted methods of bacteriological investigation.

I would urge strongly a great extension of the bacteriological methods which are employed in the investigation of material which is suspected of connexion with food-poisoning outbreaks and in the examination of dejecta from the sufferers. The close resemblance of the outbreaks in respect to clinical symptoms and signs suggests a single causative agent, but these are not infrequently present in gastro-enteritis from other causes. Savage and others have drawn attention to the importance of existing morbid conditions in determining a fatal issue, and it is worthy of consideration whether other factors may not influence the incidence rate in food-poisoning outbreaks. For example, it has been suggested that the action of chemical reagents used in pickling, such as potassium nitrate, may favour infection by the production of nitrites in the intestine. It is not unlikely that in many individuals the organisms fail to find a lodgement upon the intestinal mucosa, and little or no lesion results, although the intestinal contents contain the infecting organism.

It is remarkable how few cases in any outbreak are attended by a fatal issue, and pathological data from post-mortem examinations are correspondingly meagre. The clinical symptoms point to the upper intestinal tract as the area most affected, and this is in accordance with the findings at necropsy. The severe vomiting and purging must remove much of the unabsorbed toxic material within the alimentary canal, and the rapid recovery in many cases is presumably the beneficial result of these excretory processes. It would be expected that the cases presenting evidence of infection with living organisms would show more prolonged symptoms than in those in which only toxins are present, but in most cases recovery occurs rapidly, and evidence of invasion of the blood stream by organisms is seldom obtained. Nevertheless, the development of agglutinins to salmonella organisms is frequently reported, even in cases in which toxins only are supposed to have been present. As it is the general experience of bacteriologists that it is extremely difficult to produce antibodies in the blood of animals by administering organisms by the alimentary tract, and is only partially successful when enormous doses are given, and frequently only after starvation or in association with the feeding of agents which interfere with normal digestion, this finding of agglutinins in the blood of food-poisoning cases is the more remarkable and worthy of fuller investigation on experimental lines.

The production of agglutinins in the blood is a common occurrence after the "parenteral" injection of living or dead bacteria into the tissues of animals, but their development after a single administration by the mouth seems extremely unlikely. The presence of agglutinins in the blood of cases harbouring viable organisms is to be expected, since the organisms have the power to penetrate the mucosa and enter the deeper tissues. Their presence in the blood seems to me to be an argument against the view that only preformed toxins have been present in the offending food, and to suggest the presence of living organisms, although these may not have been detected in the course of the investigation of the outbreak. Savage and Bruce White, however, record the development of agglutinins in rabbits as a result of feeding boiled cultures of *B. aertrycke*, and Geiger and Meyer after feeding filtrates to mice and to a *Macacus rhesus* monkey. The development of agglutinins in rabbits after repeated injections of extracts of suspected meat in which no living salmonellas could be found has also been used by Savage and Bruce White, and recently also by Pryer, as a means

of demonstrating the nature of the food poisoning. The agglutinating titre of the serums which they have obtained in this way is so low and obtained with such difficulty that the test may be regarded as of little value. The possibility that such injections may merely stimulate the mechanism of agglutinin formation developed in response to a previous natural salmonella infection has apparently not been considered. There is also a lack of important control experiments—namely, the injection of similar extracts made from sound meat into rabbits of the same batch as those under experiment.

Much of the interest in the subject of food poisoning lies in the question of the production of heat-resistant poisons by organisms of the salmonella group. Gärtner in 1888, in his original studies, described the occurrence of lesions in mice after feeding meat previously infected with *B. enteritidis* and cooked for one hour. Of recent years Savage has maintained that *B. aertrycke* possesses potent heat-resistant toxins, and that these are capable of absorption in the stomach and upper intestine of man, giving rise to the phenomena of food poisoning, and that the explanation of many of the outbreaks is to be found in this property. Savage and Bruce White produced experimental results in animals which they believe support this opinion. The enormous numbers of organisms which they used in these feeding experiments produced only slight lesions in experimental animals, and it is impossible to believe that any portion of meat producing meat poisoning in man could contain such numbers. Experimental animals do not appear to be highly susceptible to feeding with salmonella cultures which have been killed by boiling or steaming, and unless we suppose that man is extremely susceptible to small amounts of such toxins these experiments fail to convince.

Other workers have also endeavoured to establish the existence of heat-resistant toxins by animal experiments. Bahr and Dyssegaard, in a recent paper, have investigated a great variety of strains of paratyphoid-Gärtner organisms isolated from a variety of sources, and were able to show that heat-resistant toxins were produced which were capable of producing severe lesions in rabbits, dogs, guinea-pigs, and monkeys, when these were injected into the tissues, but that no effect was produced by feeding even large doses by the mouth. These workers differ from Savage and Bruce White in finding that there was no relation between virulence and toxicity. Dack, Carry, and Hannon, using heat-killed cultures and filtrates of five strains of *B. aertrycke* and four strains of *B. enteritidis*, reported entirely negative results when these were fed to men on an empty stomach, and that no agglutinins had developed in the serum ten days after feeding. The preparations used by these observers were highly toxic to rabbits on intravenous injection. Young cultures of a few hours' incubation were more toxic than older cultures, whereas Bahr and Dyssegaard were of opinion that old cultures (seven to sixteen days) were most toxic. Inconclusive experiments are also reported by Branham, Robey, and Day.

On the other hand, Geiger and Meyer describe the production of a potent toxin with a particular strain of *B. aertrycke* after three to four days' incubation at 37° C. This poison was present in heated whole cultures and in filtrates, and acted by feeding as well as by parenteral injection in white mice. A special medium containing a large amount of protein was used for the cultures. The symptoms occurred within three hours, and death resulted in six to twenty-four hours. Post mortem the duodenal loop was deeply congested and moist, the stomach distended with bile and blood-streaked mucus, and petechia or small erosions were present. The spleen was enlarged, and in some cases the pleural cavities contained blood-tinged fluid. They express the opinion that these post-mortem appearances are pathognomonic for experimental food poisoning in mice. A successful experiment was also obtained with a dose of 10 c.cm. administered to a *Macacus rhesus* monkey, which developed agglutinins to a dilution of 1 in 640 two weeks after feeding. Rabbits, guinea-pigs, and cats usually presented very transitory symptoms. It would appear that mice are the most suitable animals

for this type of experiment, but it is remarkable that such difficulties should be encountered in producing toxic manifestations in animals which are frequently the subjects of natural infection with bacterial invasion of the tissues, whereas in man toxic symptoms are prominent and invasion of the tissues rarely found. It is obvious that a comparison of virulence based on the respective weights of the animals concerned is impossible, and that extreme susceptibility of the human species to the action of these toxins must be invoked in order to explain the phenomena.

It would seem a retrograde step to suggest that the difference between the experimental observations in laboratory animals and the results in man may be due to the nature of the medium in which the organisms are growing, as this would raise afresh the question of toxic substances of chemical character. It is, however, worthy of note that many of the food-poisoning outbreaks in man have been associated with tinned foods in which prolonged action of organisms may occur, and that in most of these cases no viable organisms of the salmonella group have been isolated. It is a question for consideration whether in the special circumstances existing in tinned foods these, or other, organisms, may cause the production of toxic substances which have not been obtained in artificial cultures. As the majority of laboratory animals appear to be relatively insusceptible, the experiments, particularly feeding experiments with the suspected meat, should be extended to the monkey, since Geiger and Meyer have reported a successful experiment in this animal with cultures made in a medium of high protein content.

The clinical aspect of food poisoning also presents some interesting material for speculation and investigation. As a result of a survey of epidemics of paratyphoid fever in man Savage and Bruce White have come to the conclusion that the typical signs and symptoms of food poisoning are always absent in this disease. While this is true in most outbreaks, cases are recorded in some outbreaks in which acute symptoms, diarrhoea, sickness, and abdominal pain have occurred immediately after ingestion of the infected food. The number of such cases is small, but I cannot agree with Savage and Bruce White that they may be dismissed as unimportant. For example, in the outbreak of paratyphoid fever recorded by Hamburger and Rosenthal 14 cases with symptoms of acute gastro-enteritis occurred in an outbreak affecting 65 people. Nine of these cases showed pyrexia with general toxic symptoms and intestinal catarrh, and 5 had diarrhoea with slight abdominal disturbance. In some of the cases showing typical enteric fever at a later stage slight pyrexia and vague abdominal signs were present within a few hours of infection, followed by a period of intermission before blood infection and characteristic typhoid infection declared themselves. Hamburger and Rosenthal express the opinion that the cases of gastro-enteritis were those in which little or no infection of the lymphatic tissues occurred, with resulting absence of blood infection.

In other epidemics also a few of such anomalous cases have occurred, and it is open to question whether a rigid separation of food poisoning from paratyphoid fever is justifiable on clinical grounds. Owing to early development of symptoms such cases are apt to make a powerful impression on the clinician, who finds later that enteric fever has apparently been grafted on a food-poisoning outbreak. The pathogenicity of *B. suispestifer* strains for man is generally regarded as low, since it is seldom recorded as the cause of human infections, although Scott has recently reported the occurrence of *B. suispestifer* (Group 2) in a number of outbreaks of food poisoning. On the other hand, cases with signs and symptoms of enteric fever attended by fatal issues have also resulted from infection with organisms of the *B. suispestifer* group, as in the cases recorded by Duncan and recently by Bauer and McClintock. We thus have organisms of closely related type producing symptoms of acute food poisoning, and, exceptionally, prolonged pyrexia of enteric type.

From many points of view the outbreak of food poisoning due to *B. aertrycke* recorded by Perry and Tidy is important. It embraced a large number of cases among our troops in France, and was very thoroughly examined. Although there was no direct proof, the authors were of

opinion that bacterial contamination of food by a "chronic carrier" was the cause of the initiation of the outbreak, and that the occurrence of subsequent cases was due to the usual factors of contact in a community in which acute carriers were numerous. In a selected series of 44 cases it was found that only 50 per cent. had ceased to excrete the organism by the end of the fourth week, that 2 were still positive after seven weeks, and one case positive after fourteen weeks. In two fatal cases the organism was recovered from the bile and spleen. The records of this outbreak do not lend support to the view that human carriers may not be an important source of infection. In civil life, owing to many different factors, the outbreaks are rarely examined in the same satisfactory fashion.

The laborious analysis of the salmonella group which has been carried out by Bruce White by the method of receptor-analysis has added greatly to our knowledge of these organisms, and has enabled him to correlate many apparently discordant bacteriological findings. The outstanding feature of this work from the food-poisoning aspect is the demonstration that the great majority of food-poisoning outbreaks in which organisms have been isolated are due to *B. aertrycke*. The problem of the source of infection of food materials still remains obscure, and extensive bacteriological investigations of animals and birds in contact with man may require to be carried out before satisfactory explanations are forthcoming.

The classification of the salmonella group which Bruce White has put forward shows an intimate relationship between the various members with regard to their antigenic structure which it would be out of place to discuss here. It is, however, permissible to say that there is in this work no satisfactory explanation why variants closely allied in antigenic structure should vary markedly in their toxic and invasive properties in animals. It would be a great advance in knowledge if these properties of individual strains could be definitely correlated with their antigenic constitution. The extension of this work to the field of active immunity against salmonella infections in animals is of the utmost importance towards our understanding of the phenomena of bacterial food poisoning in man.

BIBLIOGRAPHY.

- Andrewes: *Journ. Path. and Bact.*, 1922, 25, 505. *Ibid.*, 1925, 28, 345.
Savage and Bruce White: Medical Research Council, Special Report Series, 91, 92, and 103.
Doyle: *Journ. Comparative Path.*, 1927, 40, 71.
Fryer: *Amer. Journ. Public Health*, 1929, 19, 208.
Geiger and Meyer: *Proc. Soc. Exper. Biol. and Med.*, 1928, 26, 91.
Bahr and Dysegaard: *Acta Path. et Microbiol. Scan.*, 1928, 5, 116.
Dack, Carry, and Hannon: *Proc. Soc. Exper. Biol. and Med.*, 1928, 26, 49.
Branham, Robey, and Day: *Arch. Path.*, 1928, 5, 742; and *Journ. Bact.*, 1928, 15, 36.
Savage and Bruce White: *Journ. of Hygiene*, 1925, 24, 37.
Hamburger and Rosenthal: *Deut. Arch. f. klin. Med.*, 1918, 125, 415.
Scott: *Journ. of Hygiene*, 1926, 25, 398 and 406.
Duncan: *Ibid.*, 1924, 22, 402.
Bauer and McClintock: *Journ. Infect. Dis.*, 1929, 44, 292.
Perry and Tidy: Medical Research Council, Special Report Series, 24.

THE PRESENT POSITION OF THE VOLUNTARY BOARDER.*

BY

HENRY YELLOWLEES, M.D.GLAS., F.R.C.P.ED.,
PHYSICIAN AND LECTURER IN PSYCHOLOGICAL MEDICINE,
ST. THOMAS'S HOSPITAL, LONDON.

WHILE our subject cannot claim to be one of any great scientific or clinical interest, it is certainly one of the very greatest practical importance. It is a social, legal, and medical problem which merits, and will repay, the fullest discussion.

As you probably all know, there are three classes of "mental" institutions in this country: the county or borough establishments for rate-supported patients, the privately owned "licensed houses," and the registered mental hospitals. In addition to the rate-aided patients for whom they primarily exist, many of the county asylums can, and do, admit paying patients at a low figure, but no patient can be admitted to these institutions unless he has been certified and a magistrate's order for his "deten-

tion" obtained. This, by the way, I regard as probably the greatest blot on our present lunacy administration.

The licensed houses and the registered hospitals may, and do, receive voluntary boarders. A voluntary boarder is a person who enters the institution on his own request, and is free to leave it at twenty-four hours' notice should he desire to do so. We shall require, however, to look into this apparently simple definition with a great deal of care. As it stands it is not quite complete. We must add the all-important statement that a voluntary boarder may only remain under care so long as he is definitely desirous of doing so and capable of expressing his desire. In other words, expressed willingness to remain is the sole criterion of a patient's suitability to stay in a mental hospital on a voluntary basis.

This is not the law, but it is the practice, and the only thing to be said in its favour is that it is at least an improvement on the law. I think that nowadays all of us, except perhaps the more hysterical of the reformers, realize that a very profound degree of mental disturbance is quite compatible with a wish to remain under care, but in practice this is not considered. So long as the patient wishes to stay in hospital he remains a voluntary boarder, and therefore a legally sane man, whatever his symptoms may be. Once let this willingness to remain—which, by the way, may itself be a "mental" symptom—falter, disappear, or fail to be expressed, and the patient must be certified as of unsound mind. No doubt his other symptoms warrant it, and no doubt his lack of expressed willingness to remain will not appear on the certificate, but it is the crucial point nevertheless. I know of no more pitiable *reductio ad absurdum* than this, that the deciding factor as to a patient's legal sanity or insanity is simply whether or not he can express a desire to remain in hospital.

Let us consider what it implies. First, of course, it takes for granted that the patient means what he says, and will not say what he does not mean. This, no doubt, is as it should be, because it must be remembered that his mental state, medically, has nothing to do with the matter. In law he is not a lunatic; he is not even an "alleged" lunatic. Therefore his statements on the subject of willingness to remain are to be taken as the considered utterances of a man enjoying all the rights and privileges of a healthy citizen, and are to be acted on as such utterances deserve.

Now if such a patient expresses a wish to go, it would seem obvious that he must be allowed to do so. Having entered a hospital by his own desire, he must be free to leave when he so chooses, and it is unthinkable that anyone should have the power to say him nay. That sounds well, but before approving it as obvious common sense let us remember that it would hardly sound so well in the case of, say, a fever hospital. What, in fact, does the superintendent of such an institution do when, as very frequently happens, a patient in an infectious state desires to leave? He detains him against his will; he is applauded for so doing as a conscientious public servant, and he would get into serious trouble if he acted otherwise.

Passing from that, which gives considerable food for thought, we come to the bigger and more important question of the patient who, having entered a mental hospital of his own accord, becomes, owing to illness, incapable of expressing his wishes one way or another. It is this problem of the non-volitional case which has always been the centre of controversy, and has led to the familiar criticism that if a patient, fearing an acute mental attack, enters a mental hospital he will not be permitted to remain when the very illness for which he sought care supervenes, should it render him incapable of expressing his wishes.

This is perfectly true. We must note, too, that the cause of the patient's lack of volition has nothing whatever to do with the case. During all my experience as a mental hospital physician I was fortunate in having very little difficulty in the matter we are discussing, but on one occasion I had to have a voluntary boarder certified simply and solely because the old lady had a cerebral haemorrhage. Should a patient suffering from the nervous and mental after-effects of, say, concussion seek admission, and subsequently lapse into unconsciousness, there is not the

* Read in opening a discussion in the Section of Neurology and Psychological Medicine at the Annual Meeting of the British Medical Association, Manchester, 1929.