found that 5% of 400 girls examined in a remand home for juveniles aged 14–17 were prostitutes at the time of their arrest, and another 5% of 200 who were followed up took to prostitution in the next few years. Nearly all the girls were sexually experienced or promiscuous and often "on the run." The rarity of prostitution in such wayward girls in itself points to the existence of some emotional predisposition. Three groups were distinguished. The largest was composed of highly intelligent (average I.Q. 125) but very unstable girls; half of them attempted suicide at some time, and many of them had overt homosexual interests. Their background had been extremely traumatic, leaving a tendency to hostility and resentment which disturbed all their relationships. A feature common to all the prostitutes was that they had a thinly veiled hostility to men and fear of being dominated by them. This was also a feature of other girls who seemed in danger of future prostitution—mercenary girls whose lack of feeling allowed them to be contemptuous gold-diggers, and girls who associated with coloured men because they found them less possessive and dominating than white men. Menstrual irregularity, which is rare in wayward girls, was also a feature of the prostitutes. When these young prostitutes were followed up, it was found that most of them gave up prostitution quite readily and many had successful and valuable careers; several became nurses. This raises the question whether prostitution is not usually, like crime in men, a transitory event. Kinsey[4] insists that "a very high number of the females who engage in such activities do so as a minor adjunct to their regular occupations." The important problem may be why some persist.

Social forces cannot fail to be important in prostitution, and they are emphasized by Mrs. Wilkinson[5] in her study of London prostitutes. Her most valuable contribution to the psychology of the prostitute concerns the ponce, whom she shows to be actively chosen by the prostitute and faithfully maintained in spite of quarrels and prison sentences. The procuration of prostitutes, like the seduction of homosexuals, is now regarded as of less aetiological importance than used to be thought. It is clear too that, just as the homosexual prostitute is not always very strongly homosexual, the sexual predisposition of the prostitute may not need to be strong to make her succumb to social pressures. Prostitutes have a strong feeling of "us girls" and form a subculture which thrives on the disapproval of society. In this subculture a girl can steal from her best friend and cheat her of her love-partner. This lack of persistent relationships seems elevated to a principle, and it must offer a haven of refuge to those who are incapable of normal or persistent relationships.

The existence of intellectually brilliant young prostitutes is a reminder that throughout history many famous women have been prostitutes or near-prostitutes. The passive, receptive sexual role, and the conventionality which our society imposes upon women, make it difficult on the one hand to detect the nature of their sexual perversions and on the other hand to examine dispassionately the phenomenon of female rebellion or originality. The scores of books on prostitution are, as Kinsey remarks, out of proportion to its sexual, if not its social, significance. With the increasing awareness of the intimate interaction of culture and personality in its production, some progress may be made. And it is increasingly apparent that this will include a close study of the prostitute's client, who keeps up the demand.

TROPHIC LESIONS IN NERVOUS DISEASE

Trophic lesions in the limbs and extremities may result from a variety of lesions of the nervous system. In patients with long-standing disease of the lower motor neurone (as a consequence, for instance, of poliomyelitis) the affected limb may become blue and cold and sometimes swollen, while the skin eventually becomes shiny and atrophic. When the sensory pathways, and particularly those subserving pain sensation, are also affected, trophic lesions may be much more severe, giving rise to chronic ulceration and in certain cases to gross disorganization of bones and joints with extensive mutilation. In all disorders of this nature it is still doubtful how much of the destruction results from repeated painless trauma and how much from loss of nutrition of the tissue owing to damage of autonomic fibres and, secondarily, of blood supply. The peripheral ulceration and deformity seen in the neuropathic form of leprosy is all too familiar in countries where the disease is endemic, while a condition more familiar in Great Britain is the insensitive, scarred, and brawny hand, with perhaps a Charcot's arthropathy at the elbow or shoulder, seen in patients with chronic syringomyelia. In tabes dorsalis, too, perforating ulcers of the soles of the feet and swollen, flail joints in the lower limbs are common. Severe destructive changes of this degree are rare in sporadic cases of polyneuropathy, but here too the hands and feet may be cold, brawny, and swollen and may sweat profusely, while in cases of peripheral nerve lesions, such as severe compression of the median nerve in the carpal tunnel, similar skin changes and even ulceration may occur in insensitive areas.

Less well known is the curious group of disorders to which Dr. L. van Bogaert draws attention in the opening pages of the *Journal* this week. In these the clinical

[1] Denny-Brown, D., *J. Neurol. Neurosurg. Psychiat.*, 1951, **14**, 237.
[2] Munro, M., *British Medical Journal*, 1956, **1**, 541.
[3] Ibid., 1957, **1**, 510.
[4] Jewesbury, E. C. O., *Brain*, 1951, **74**, 336.
[5] Refsum, S., Heredopathia Atactica Polyneuritiformis, Tanum, Oslo, 1956.
[6] Andrade, C., *Brain*, 1952, **75**, 408.
[7] Denny-Brown, D., and England, A. C., *A.M.A. Arch. Neurol. Psychiat.*, 1952, **67**, 1.

picture is suggestive of a lumbosacral syringomyelia, often occurring in more than one member of a family. Among the many titles used for this group of conditions are Morvan's syndrome, hereditary perforating ulcer of the foot, neurotrophic atrophy, and acro-osteolysis. In many cases of this type the disease gives rise to one or more perforating ulcers of the feet, with blueness and coldness of the limbs, and this may be followed by extensive ulceration, leading to bony destruction and mutilation. On examination the clinician usually finds extensive peripheral loss of pain and temperature sensibility, affecting the lower limbs particularly, but often the hands and arms as well. Whereas in certain cases of this type histological studies have shown cavitation in the central areas of the lumbar spinal cord, like that of syringomyelia, or else an anomaly of cord development or myelodysplasia resulting from incomplete closure of the neural tube, the work of D. Denny-Brown[1] has suggested that in most such cases the primary lesion is in the posterior root ganglia and that the condition is best referred to as "hereditary sensory neuropathy." When occurring in a young child the condition may present as an apparent congenital indifference to pain,[2] which was discussed in these columns earlier this year,[3] but testing will reveal that the insensitivity is peripheral in distribution, unlike the rare cases of "pain asymbolia" which are relatively unaffected by painful stimuli applied to any part of the body.[4] The fact that deafness is almost always found in patients with sensory neuropathy suggests a close relationship between this disorder and the "heredopathia atactica polyneuritiformis" of S. Refsum.[5] Since Morvan's description in 1833 was of a disease occurring in a group of fishermen in Brittany, it is of interest to note that C. Andrade[6] has recently described a form of peripheral neuropathy, shown histologically to be due to primary amyloidosis and giving trophic lesions and pain loss in the lower limbs, which was endemic in the fishing village of Povoa de Varzim in Portugal. In other words, there seem to be many different degenerative neurological syndromes which differ in detail but have as a common feature genetic determination, perforating ulcers of the feet and other trophic lesions leading to mutilation, and peripheral sensory loss affecting principally pain and temperature sensation. The fact that changes of this type occur in certain families with peroneal muscular atrophy[7] and Friedreich's ataxia suggests that hereditary sensory neuropathy may be another of those hereditary degenerative disorders of the nervous system which may sometimes occur alone in pure form, but may on other occasions be combined with other member diseases of the "hereditary ataxia" group in various combinations.

Finally, there are the disorders believed to be due to disturbances of the autonomic nervous system which give rise to trophic lesions in the extremities. The swollen, cyanosed, and burning hands and feet of the infant with pink disease are well known to every paediatrician; while in some cases the condition is attributable to mercury poisoning, in others the nature of the neuropathic disturbance remains unknown. Little, too, is known about the aetiology of causalgia, of the post-traumatic Raynaud phenomenon, and of the shoulder–hand syndrome, though the relief experienced in each of these conditions after sympathectomy suggests that a disturbance of the autonomic nervous system is responsible. Causalgia takes the form of an intense burning pain and hyperaesthesia in the distribution of a peripheral nerve (usually the median or sciatic), and usually follows an injury to the nerve. The post-traumatic Raynaud phenomenon generally follows a non-specific injury to the upper limb or else occurs in workers who use vibrating tools; the hand may become cold, swollen, hyperhidrotic, and intensely painful. The condition is closely related to the shoulder–hand syndrome, in which a similar painful swelling of the hand is associated with a pericapsulitis of the shoulder-joint. The latter condition, which may follow upon trauma or cardiac infarction, may, if severe, result in atrophy and decalcification of bones of the extremity (Sudeck's atrophy). Clearly much remains to be learned about these curious conditions. Indeed, the entire range of trophic disorders occurring in neurological disease and their prevention are worthy of study, for they may be a serious cause of disability.

FALSE ADMISSIONS OF PATERNITY

Most lawyers in Great Britain are well aware nowadays that paternity can be positively excluded if the putative father's blood group is genetically incompatible with that of the child. An unusual case heard recently in the Scottish Court of Session is reported in our medico-legal column this week (p. 417). But magistrates cannot compel a mother in affiliation proceedings to submit herself or her child to a test, and in consequence the court often has to come to a decision on incomplete evidence. However much the putative father may ask for a test, which, if he is falsely accused, will give him something like a 50% chance of conclusively clearing himself, the court cannot order one to be made. In practice no court draws an inference adverse to the mother if she refuses to submit to a blood test for herself and her child when invited by the putative father's advisers to do so.

A recent report[1] from the U.S.A. points to some anomalies, which might also be found in Britain. L. N. Sussman and S. B. Schatkin, of New York, persuaded the parties in proceedings for affiliation in the United States to submit themselves to blood tests after the putative fathers had admitted paternity and the equivalent of affiliation orders had been made against them. The investigators observe that obtaining the co-operation of the parties after their cases were completed was understandably difficult. Nevertheless they were able to make tests on the parties in 67 cases, and these showed that in no fewer than six cases the putative father who had not disputed paternity could not have been the father of the child. The investigators state that in the United States most men involved in paternity suits do not contest the charge, and it is only those who do so who are privileged by the law of many states to request blood

[1] Sussman, L. N., and Schatkin, S. B., *J. Amer. med. Ass.*, 1957, **164**, 249.

tests to sustain their denial. They comment that the result of their investigation shows it to be in the interests of justice that a blood test should be ordered in every case where paternity is in issue, so that even a putative father who from whatever motive is prepared to admit paternity, or is deceived into doing so, will at least not be held to be father of a child who, unless the highly improbable event of a suitable mutation has occurred, cannot be his own. They conclude that the demonstrated unreliability of admissions of paternity should be drawn to the attention of the proper authorities.

Whatever may happen in the U.S.A., no change is likely in England. When a putative father is prepared to accept responsibility for a bastard, the mother's corroborative evidence is essential here before an order can be made, whether he admits paternity or not; and, even if abstract justice requires the exclusion of such error as can be excluded by blood tests, it is difficult to imagine that Parliament in the interests of justice would interfere to make that exclusion compulsory.

SANITATION AND POLIOMYELITIS

A patient with poliomyelitis may excrete in one gramme of faeces enough virus to infect one million monkeys.[1] Half the patients are still excreting virus after three weeks, and some continue to excrete it up to twelve weeks. Since many rivers, streams, and ditches in Great Britain, as in other countries, are polluted by human excreta, it is a wise precaution to advise parents to keep their children away from them when cases of poliomyelitis are occurring in the area. Medical officers of health in many districts have found it advisable this summer to give this warning, and to emphasize that hygiene in the home may help to prevent the disease. Flies are known to be capable of carrying faecal material contaminated with virus, so that measures to destroy them and to prevent any reaching food are well worth carrying out assiduously. Yet the curious fact remains that sound evidence is still lacking that poliomyelitis is mainly a water-borne or food-borne disease, like dysentery, for instance, or food-poisoning, though a recent study[2] in the U.S.A. suggested a connexion between cases of the disease and a water supply contaminated with excreta from lavatory pans. Careful studies have shown that the virus is usually spread by close contact, and that the path of its spread, when it can be traced, is sometimes quite narrow.[3] How wide or narrow this path of infection may be is still disputed,[4] but observers seem to be agreed that poliomyelitis is what has been called a family disease. Spread of infection within a family is so rapid that when a case is detected all the other members of the household are normally found to give serological evidence of infection. Transmission beyond the bounds of the family seems to be principally by personal contact at work—as doctors in particular know[5] from the additional risks they run—or at play from child to child in gardens and streets.

As well as advocating hygiene in the home, early reporting of feverish illness, and moderation in exercise, some form of segregation of contacts is generally held to be desirable, a period of three weeks being suggested.[4] A final point worth considering is whether people should be advised to stop bathing off the shore of a seaside resort where cases are occurring. If sewage containing poliomyelitis virus is pumped into the sea and it comes into contact with bathers, there would seem to be some risk of disease being thus transmitted. No outbreak of poliomyelitis has been tracked down to this particular source, and it is probably a reasonable inference that the risk of infection, at present, is exceedingly slight. But while coastal resorts continue to grow, and more and more people take seaside holidays, England is fast becoming a jewel set in a sea not of silver but of sewage. A thorough study of this public health problem seems overdue.

[1] *Wld Hlth Org. techn. Rep. Ser. No. 81. Expert Committee on Poliomyelitis*, 1954.
[2] Bancroft, P. M., Engelhard, W. E., and Evans, C. A., *J. Amer. med. Ass.*, 1957, **164**, 836.
[3] *British Medical Journal*, 1953, **2**, 1258.
[4] Ibid., 1956, **2**, 32.
[5] Doll, R., and Hill, A. B., ibid., 1957, **1**, 372.

PINK DISEASE

Even to those enjoying normal colour vision the aetiology of pink disease is evergreen. Presenting in infancy, of self-limited duration, and rarely recurrent, it appears to be commoner in some regions than in others. Such features as cold, pink extremities, pain, misery, trophic changes, profuse sweating, sudaminal rashes, tachycardia, hypertension, constipation, and dysuria appeared to E. Feer to be due to a disordered vegetative nervous system.[6] Others have pointed to the possible aetiological role of such insults as measles, maternal deprivation, and mercury. Impressed by some resemblances to mercurial poisoning, J. Warkany and D. M. Hubbard[7] successfully demonstrated abnormal amounts of the metal in the urine of infants with pink disease, and this has been confirmed repeatedly. But infants may ingest mercury without harm, and some may develop pink disease without absorbing mercury in any form. Some "mercurial pinks" have responded to dimercaprol, many have not, and some have responded although they excreted no mercury before, during, or after treatment. Others have successfully survived treatment with mercury before, during, and after the condition. Hypersensitivity to mercury has been postulated to explain why so few infants receiving mercurial medicines develop pink disease. Yet the infants of Warwickshire, according to A. Holzel and T. James,[8] ingested mercurial powders five times more frequently than those of Manchester, but had less than a quarter of the latter's incidence of pink disease; other,

[6] Feer, E., *Ergebn. inn. Med. Kinderheilk.*, 1923, **24**, 100.
[7] Warkany, J., and Hubbard, D. M., *Lancet*, 1948, **1**, 829.
[8] Holzel, A., and James, T., ibid., 1952, **1**, 441.
[9] Colver, T., *British Medical Journal*, 1956, **1**, 897.
[10] Cheek, D. B., and Hicks, C. S., *Med. J. Aust.*, 1950, **1**, 107.
[11] Williams, H., Macdonald, W. B., and Callow, V., ibid., 1951, **1**, 363.
[12] Cheek, D. B., *J. Pediat.*, 1953, **42**, 239.
[13] —— *Pediatrics*, 1957, **19**, 68.
[14] Hubble, D., *Arch. Dis. Childh.*, 1951, **26**, 340.
[15] Vulliamy, D. G., *Lancet*, 1952, **2**, 1248.
[16] Farquhar, J. W., Crawford, T. B. B., and Law, W., *British Medical Journal*, 1956, **2**, 276.
[17] Leys, D., *Arch. Dis. Childh.*, 1950, **25**, 302.
[18] Bower, B. D., *Quart. J. Med.*, 1954, **47**, 215.

less likely, mercurial medicines were not considered by them. Because of concern about the possible aetiological role of the metal it was withdrawn from popular "teething powders," and prescriptions have decreased. According to T. Colver[9] a rapid fall in the consumption of mercury by Sheffield infants has been paralleled by a decreased incidence of pink disease, though the fall in incidence of the disease appears to date from 1951, whereas mercury-free teething powders became freely available in 1954. A reasonable case against the use of mercury exists, however, and there are less harmful and equally efficacious remedies for the fretful or infected child.

In a paper entitled somewhat prematurely "Pink Disease, Its Nature, Prevention and Cure" D. B. Cheek and C. S. Hicks[10] recorded that deficiency of water and salt occurred in this condition, and they ascribed this to stress hypoadrenocorticism. According to them the chain of events is that water moves into the cells, with resultant cell turgor and cerebral oedema, shown by irritability and photophobia. Haemoconcentration explains the high haematocrit, raised serum protein, tachycardia, hypertension, stagnation, anoxia of the peripheral organs, and constipation. Heat regulation is impaired and compensatory sweating takes place. Administration of salt, water, and deoxycortone resulted in dramatic improvement. As the authors said, "The theory was found to apply almost suspiciously well." The electrolytic and therapeutic findings were not reproduced in Melbourne[11] or in the more temperate United Kingdom. Further work by Cheek[12] confirmed the occurrence of oligaemia and hypochloraemia and the limited value of salt and deoxycortone. He considered that steroid studies provided evidence of hyperadrenocorticism in pink disease, and he quoted experimental evidence for his belief that hypokalaemia also existed. The basic upset appeared to be the loss of water and choride, but replacement of these was not curative.

Workers investigating apparently unrelated aetiological theories may sometimes find they share common ground, and Cheek[13] has wedded his ideas to those that blame mercury. The result is attractive, and would be more so if all pink disease were undoubtedly due to this metal. He now believes that the renal tubules of the hypersensitive child are damaged by mercury; rejection of water and chloride results in an initial diuresis, and total volumes of body water and chloride are impaired. The resultant oligaemia and hypochloraemia initiate the chain of events described above. The kidney gradually succeeds in conserving water, but loses salt until spontaneous remission begins. A possible increase in metabolic rate is suggested to explain the increased sweating, which Cheek believes to be unsurpassed in any other disease. The electrolyte content of the sweat is low, as in heat stress, but the volume is so great that the total loss of electrolyte in this way is high. Some would say the sweating of phaeochromocytoma was even greater, and D. Hubble[14] and D. G. Vulliamy[15] have found that the two conditions have much in common. The clinical picture of phaeochromocytoma results from the action of sympathetic amines, and according to J. W. Farquhar and colleagues[16] they may be excreted in excess in pink disease. These authors consider the possibility of pink disease being the result of a central disorder of sympathetic control and are thus in accord with Feer[6] and D. Leys.[17] Why should the sympathetic nervous system be deranged? Here some would agree with Cheek's original conception of response to stress. Perhaps like a number of other conditions pink disease may result from the conjunction of a constitutional factor and an environmental stress. Of the latter mercury may be the most important and is best not given. Deficiency of salt and water, when present, should be remedied, but symptomatic relief of the child's distress is more likely to follow the skilful use of an autonomic blocking agent.[18]

DRUGS FOR PRIVATE PATIENTS

A Bill to amend Section 38 of the National Health Service Act, 1946, was presented by Mr. John Hall and Mr. Ronald Bell on July 31. The amendment sought reads as follows:

> "Notwithstanding anything contained in section thirty-eight of the National Health Service Act, 1946, every private patient of a registered medical practitioner shall, as from the passing of this Act, be entitled to be supplied with drugs and medicines and prescribed appliances pursuant to the said Act, on the same terms as patients who are registered with a medical practitioner who provides general medical services under the said Act."

It is satisfying to see this aspect of Association policy receiving this degree of support. The arguments in favour of allowing patients to enjoy benefits of any part of the N.H.S. are well known and need not be repeated here. Apart from any argument, the public was promised this before the appointed day. But the drafting of the Act made it possible for the politician to take refuge from an action that might be politically unsafe. Members of the Association who are especially interested in the Association's policy of free drugs for private patients can support it by urging their M.P.s to see that Mr. John Hall's and Mr. Ronald Bell's Bill gets a fair hearing.

PRINCIPAL AT BANGOR

Mr. R. C. Evans, F.R.C.S., has been appointed principal of the University College of North Wales, Bangor. One of the most experienced mountaineers in the world, Mr. Evans played a prominent part in the 1953 Everest expedition, when Hillary and Sherpa Tensing reached the summit. Two years later he led the expedition which climbed Kangchenjunga for the first time. Now aged 38, Mr. Evans qualified with the Oxford degree in 1943, and after serving in the R.A.M.C. took the F.R.C.S. in 1949. In 1956 he received the honorary D.Sc. from the University of Wales. At present Mr. Evans is working as surgical registrar with the United Liverpool Hospitals (Royal Southern Hospital). He will give up medical practice when he takes up his new duties next year. At Bangor he will have the pleasure of living within sight of the Snowdon range, where he gained his first experience of mountaineering.