Discussion

From the above evidence it may be concluded that certain "autoimmune diseases" tend to occur together in various combinations and that thyrotoxicosis also participates in combinations with these diseases. In advancing these conclusions as evidence suggesting that thyrotoxicosis is an autoimmune disease, we are well aware that, with the possible exception of certain blood dyscrasias, no pathological process has been shown to be the result of autoimmunity: the evidence is perhaps strongest, but still not conclusive, in relation to chronic thyroiditis.

Our evidence that thyrotoxicosis is an autoimmune disease is based entirely on its belonging to a group of diseases which tend to accompany one another and in which pathological changes in a particular tissue are commonly associated with the development of autoantibodies to that tissue. We are certainly not suggesting that any of the known thyroid autoantibodies is the cause of thyrotoxicosis. However, it is of considerable interest that in 1957 Adams and Purves reported that the abnormal thyroid stimulator (L.A.T.S.) demonstrable in the blood in some cases of thyrotoxicosis is present in the γ -globulin fraction of serum, while Kriss *et al.* (1964) have reported that L.A.T.S. has the characters of a 7S- γ -globulin, and have suggested that it may be an autoantibody to the thyroid. If this claim is substantiated it seems probable that thyrotoxicosis will prove to be the first organ-specific autoimmune disease attributable to a circulating antibody, for the only known biological effect of L.A.T.S. is its stimulating effect upon the thyroid of various experimental animals.

Summary

The reported high incidences of thyroid and gastric antibodies in the serum of thyrotoxic patients was confirmed, and it was shown that the two types of autoimmunity tend to occur especially in the same thyrotoxic individuals. Evidence that a genetically determined factor predisposes to various organ-specific autoimmunities is reviewed, and it is concluded, from the known occurrence of various autoimmunities in patients with thyrotoxicosis, that autoimmunization plays an important aetiological part in thyrotoxicosis itself.

ADDENDUM.—Since this paper was submitted for publication the possible autoimmune nature of thyrotoxicosis has been discussed by D. Doniach and I. M. Roitt in *Seminars in Haematology*, 1964, 1, 313.

REFERENCES

- Adams, D. D., and Purves, H. D. (1957). *Canad. J. Biochem.*, **35**, 993.
 Adams, J. F., Glen, A. I. M., Kennedy, E. H., MacKenzie, I. L., Morrow, J. M., Anderson, J. R., Gray, K. G., and Middleton, D. G. (1964). *Lancet*, **1**, 401.
 Anderson, J. R., Goudie, R. B., Gray, K. G., and Buchanan, W. W. (1961). *Scot. med. J.*, **6**, 449.
 ——— and Timbury, G. C. (1957). *Lancet*, **1**, 1123.
 Blizzard, R. M., Chandler, R. W., Kyle, M., and Hung, W. (1962). *Ibid.*, **2**, 901.
 ——— and Kyle, M. (1963). *J. clin. Invest.*, **42**, 1653.
 Bock, O. A. A., and Wirts, L. J. (1963). *Brit. med. J.*, **2**, 20.
 Buchanan, W. W., Koutras, D. A., Crooks, J., Alexander, W. D., Brass, W., Anderson, J. R., Goudie, R. B., and Gray, K. G. (1962). *J. Endocr.*, **24**, 115.
 Davidson, W. M. B., and Markson, J. L. (1955). *Lancet*, **2**, 639.
 Doniach, D., Roitt, I. M., and Taylor, K. B. (1963). *Brit. med. J.*, **1**, 1374.
 Hall, R., Owen, S. G., and Smart, G. A. (1960). *Lancet*, **2**, 187.
 ——— Saxena, K. M., and Owen, S. G. (1962). *Ibid.*, **2**, 1291.
 Irvine, W. J. (1963a). *Quart. J. exp. Physiol.*, **48**, 427.
 ——— (1963b). In *The Thyroid and its Diseases*, edited by A. Stuart Mason, p. 129. Pitman, London.
 ——— (1963c). *J. Endocr.*, **26**, XXXII.
 ——— Davies, S. H., Delamore, I. W., and Wynn Williams, A. (1962). *Brit. med. J.*, **2**, 454.
 Kriss, J. P., Pleshakov, V., and Koblin, R. (1964). *Clin. Res.*, **12**, 116.
 Long, O. A. (1957). In *Ciba Foundation Colloquia on Endocrinology*, **10**, 287, edited by G. E. W. Wolstenholme and E. C. P. Miller. Churchill, London.
 McNicol, G. P. (1961). *Amer. J. med. Sci.*, **241**, 336.
 Magnus, H. A. (1958). *J. clin. Path.*, **11**, 289.
 Markson, J. L., and Moore, J. M. (1962). *Lancet*, **2**, 1240.
 Osserman, K. E., and Silver, S. (1961). In *Advances in Thyroid Research*, edited by R. V. Pitt-Rivers, p. 100. Pergamon, Oxford.
 Roitt, I. M., and Doniach, D. (1960). *Brit. med. Bull.*, **16**, 152.
 Rupp, J. J., and Paschkis, K. E. (1957). *J. clin. Endocr.*, **17**, 143.
 Schmidt, M. B. (1926). *Verh. dtsch. path. Ges.*, **21**, 212.
 Simpson, J. A. (1964). *J. Neurol. Neurosurg. Psychiat.* In press.
 Sloper, J. C. (1953). *J. Path. Bact.*, **66**, 53.
 Smith, A. W. M., Delamore, I. W., and Wynn Williams, A. (1961). *Gut*, **2**, 163.
 Stewart, W. K., Green, D. M., and Lowe, K. G. (1962). *Acta endocr. (Kbh.)*, **40**, 613.
 Taylor, K. B., Roitt, I. M., Doniach, D., Couchman, K. G., and Shapland, C. (1962). *Brit. med. J.*, **2**, 1347.
 te Velde, K., Abels, J., Hoedemaeker, Ph. J., Arens, A., and Nieweg, H. O. (1964). *Lancet*, **1**, 941.
 Tudhope, G. R., and Wilson, G. M. (1960). *Quart. J. Med.*, **29**, 513.
 ——— (1962). *Lancet*, **1**, 703.
 Wells, H. G. (1930). *Arch. Path.*, **10**, 499.
 Wilkinson, J. F. (1949). *Lancet*, **1**, 336.
 Williams, E. D., and Doniach, I. (1962). *J. Path. Bact.*, **83**, 255.

Low Incidence of Latent Trichinosis Near Blackpool Compared with Incidence Elsewhere in England and Wales

P. W. HARVEY,* V.R.D., M.B., M.C.PATH.; W. E. KERSHAW,† V.R.D., M.D., D.SC.

Brit. med. J., 1964, **2**, 1632–1634

Outbreaks of trichinosis are rare in England and Wales compared with Europe and the U.S.A. For each outbreak, however, there must be many latent cases associated with those that are diagnosed, and there must also be other sporadic infections occurring independently of these outbreaks. Outbreaks in which the parasite was demonstrated have occurred in Workington (1874), Thaxted, Essex (1879), Swansea (1930), Haverfordwest (1939), Wolverhampton (1941), Harpenden (1941), Penrith (1941), Birmingham (1941), Lewisham (1941), Barry, Glamorganshire (1952), and Liverpool (1953).

* Lately Consultant Pathologist, Victoria Hospital, Blackpool. Now at the Department of Pathology, Royal Infirmary, Lancaster.

† Professor of Parasitology and Entomology, Liverpool School of Tropical Medicine and University of Liverpool.

The outbreak in Wolverhampton provoked an inquiry by May Young (1947) on the incidence of latent trichinosis in England and Wales, and she carried out an investigation on cadavers in Birmingham, Wolverhampton, Bristol, Llandough, Cardiff, and Leeds. The Liverpool outbreak in 1953 reopened the question of the incidence of latent infections.

Outbreaks of infection are focal and isolated; even in 1941, the second year of the war, when meat production and distribution had altered, there was no apparent connexion between the separate outbreaks. It seemed to us that the latent infections might also be focal, and that the "average incidence of 10%" in the population in the United Kingdom was spurious. For most cities, and certainly for Liverpool, the pork is produced elsewhere, and in most rural areas in this country it is produced

and consumed locally. We therefore examined the incidence of latent infections in a rural district near Liverpool (Blackpool and the Fylde) where we knew the source of pork to be local.

The seaside towns near Blackpool form a continuous built-up area along the coast from Fleetwood to Lytham, with the rich agricultural land of the Fylde behind it. These towns are involved mainly with the seasonal holiday trade, although there are a few new factories. Many of the permanent inhabitants came on retirement; about 1,200 pensioners come to live in Blackpool each year. The total population of the borough is 150,000. There is also a constant stream of younger people coming to try their hand at the boarding-house trade, but it is impossible to estimate their numbers; some stay, but many move out again after a few years and are replaced by others. Most of the people who come to the area either on retirement or to work are from industrial Lancashire, particularly from south-east Lancashire; the others are from Yorkshire and the Midlands. Only approximately one-third of the town council are natives.

Latent trichinosis in the Blackpool area might be due either to old infections acquired before the individuals concerned arrived there or to new infections acquired after arrival. Most of the pork products consumed along the coast are made from animals which have been reared on farms in the area, where pig-farming is a thriving industry.

The present survey was begun in 1958 and completed in 1963. It was started after experimental work on the transmission of trichinosis had been suspended, so there is no question of contamination in experimental procedures.

Methods

The cases were, apart from the exclusion of children, unselected routine necropsies carried out by one of us in Victoria Hospital, Blackpool. Twenty grammes of muscle were taken from the diaphragm and 20 g. from the lateral aspect of the thigh (Kershaw, St. Hill, Semple, and Meredith-Davies, 1956). The post-mortem examinations were usually made within a day of death, and two sites were chosen in order to increase the probability of finding infections of very low intensity. Material to be digested was posted to Liverpool within two days, and frequently within one day, of the post-mortem examination.

Specimens were digested overnight in 1% pepsin at body temperature, and the whole of the deposit was mixed with formalin and then examined for larvae and cysts in square-rimmed Petri dishes under a low-power microscope.

Results

Larvae of *Trichinella spiralis* were found in two cadavers of the 512 that were examined; the overall incidence was thus 0.4%. One of the two individuals was a regular soldier aged 59 who had served a long time abroad; the examination showed two larvae in the thigh and one larva in the diaphragm. The other was a woman aged 65 who came from Barrow-in-Furness, not far from Workington; the examination showed five larvae in the diaphragm but none in the thigh. The age distribution of the individuals examined is shown in Table I.

In 1845 latent trichinosis was estimated in Edinburgh, presumably by direct examination rather than by digestion, by Knox as 1 in 100, by Handysides as 0 in 143, by Lizars as 1 in 200–300, and by Mackenzie as 3 in 500; Turner (1860)

estimated the incidence as 1–2% in five years' necropsies. Table II shows, on a geographical basis, the information available from the surveys of van Someren (1937), Jolly (1943), and May Young (1947), together with that obtained from our own survey during 1958–63.

TABLE II.—Recorded Incidences of Latent Infections With *T. spiralis* in Man in England and Wales

Locality	No. of Cadavers Examined	Cadavers Infected	
		No.	%
1947 Birmingham	194	24	12.4
1947 Wolverhampton	106	16	15.1
1943 Black Country	56	10	17.8
1947 Bristol	48	6	12.5
1947 Cardiff	56	2	3.6
1947 Cambridge	37	2	5.4
1947 Llandough	7	1	14.3
1947 Leeds	24	0	0
1937 London	200	2	1.0
1958–63 Blackpool	512	2	0.4

Discussion

It is difficult to assess the significance of the latent infections observed in Bristol, Cardiff, Cambridge, Llandough, and Leeds because of the small numbers examined and the small numbers of positives found, but in Wolverhampton, where an outbreak had occurred, the incidence was 15% and in Birmingham it was 12%. The incidence which we have observed in the Blackpool area is less than 1%.

It is possible that the incidence in Birmingham and Wolverhampton is abnormally high compared with the rest of the country, and that the incidence in parts of the country such as Blackpool is very low.

It is also possible that Birmingham and Wolverhampton represent an area, rather like Liverpool, where outbreaks are likely to be diagnosed because the risk of infection may be high and there is an awareness of this possibility.

Finally, it is possible that the incidence generally has fallen over the last 20 years; but although Knox (1845) carried out his investigation over 100 years ago, Turner (1860) his about 100 years ago, and van Someren (1937) his 27 years ago—all in localities where no outbreak has ever been established—their figures are of the same order as ours.

May Young (1947) attempted to draw an average incidence of infection with *T. spiralis* in this country: we think her method is invalid and her average therefore unreal. She says that the average infection rate in England is about 10%; we consider this meaningless. As a corollary, we believe that Miss Madsen (1961), who cited May Young's "average" of 10% as being reprehensible in a civilized country, was wrong in criticizing the incidence of trichinosis in Britain as being high.

We therefore believe that to elucidate the state of latent trichinosis in England requires the review of many areas where this latent infection has been assessed separately and where it also has been related to the risk of infection as revealed by diagnosed outbreaks.

Summary

The incidence of latent trichinosis in 512 routine necropsies examined by digestion was 0.4% in an area near Blackpool. Comparison of these results with such other investigations as have been done in other places in England and Wales shows that the distribution of latent trichinosis is very uneven. Before attempting to define the epidemiology of this infection many more surveys must be done among communities of different composition and having different sources of meat supply.

This investigation was supported in part by a grant from the Research Fund of the Manchester Regional Hospital Board.

TABLE I.—Age Distribution of 512 Cadavers Examined for Infection With *T. spiralis*

Age in years	<11	11–	21–	31–	41–	51–	61–	71–	81+	Not Noted
No. . . .	0	7	14	28	70	136	162	81	9	5

REFERENCES

- Handysides, P. D. (1845). *Lond. med. Gaz.*, **32**, 805.
 Jolly, R. H. H. (1943). *Med. Offr.*, **69**, 13.
 Kershaw, W. E., St. Hill, C. A., Semple, A. B., and Meredith-Davies, J. B. (1956). *Ann trop. Med. Parasit.*, **50**, 355.
 Knox (1845). *Lond. med. Gaz.*, **32**, 805.

- Lizars, A. S. (1845). *Ibid.*, **32**, 805.
 Mackenzie (1845). *Ibid.*, **32**, 805.
 Madsen, H. (1961). *Medd. Grønland*, **159**, No. 7, p. 1.
 Turner, W. (1860). *Edinb. med. J.*, **6**, 209.
 van Someren, V. D. (1937). *Brit. med. J.*, **2**, 1162.
 Young, May R. (1947). *J. Helminth.*, **22**, 49.

The Drinking Driver

SIMON FREEMAN,* O.B.E., T.D., L.R.C.P.S.ED., L.R.F.P.S.GLAS.

Brit. med. J., 1964, **2**, 1634–1636

A period of six years' experience in Manchester is covered in this survey. It includes the first 100 cases already described (Freeman, 1960). During this period a total of 392 persons were seen who had been apprehended by the police on suspicion of being under the influence of alcohol while in charge of a motor vehicle, and, though the clinical picture remained consistent throughout, several features emerged with greater clarity. A disturbing trend was the progressive increase in the number of drivers brought in for examination, and this appeared to be in line with conditions throughout the country. There was no consistency in the pattern of incidence save for the annual pre-Christmas "bulge." Because speed in calling the police medical officer had been emphasized, most of the arrested drivers were examined within 20 minutes of their arrival at the police station.

It was made clear to every driver at the outset that he had the right to refuse to be examined. Refusal was almost invariably associated with drivers manifestly certifiable as being intoxicated, and it was evident throughout that the less alcohol a driver had consumed the more ready—and even anxious—he was to agree to examination, while the more he had imbibed the quicker he was to clutch at the straw of refusal. It would be advisable to make it less simple to refuse to be examined, and we might well copy the method employed in New York State where application forms for driving licences contain a paragraph giving the police permission to order the necessary examination in such cases. Refusal is still permitted, but in that event the licensing authorities are immediately notified and the licence revoked.

Examination

Once the driver had given his permission and read and signed a form of consent he was examined along the lines laid down in *Recognition of Intoxication* (British Medical Association Special Committee, 1958). In arriving at an opinion a careful assessment had to be made of the overall picture, making full allowance for worry, fatigue, and excitement, together with all tests satisfactorily performed. Above all, one always had to bear in mind the oft repeated axiom: "With the exception of the smell of the breath each and every symptom or sign can be explained by a cause other than alcohol." While no single sign could conceivably be decisive in arriving at an opinion, the following signs appeared together with such consistency that their presence together was regarded as of the greatest significance diagnostically: (1) slurred speech; (2) full, bounding pulse; (3) impaired memory; (4) poor co-ordination; (5) widely dilated pupils with very little or no reaction to strong light—this sign presented itself with such consistency that it was considered to be one of the most suspicious and significant signs, and, indeed, one which cannot be "faked"—and this fact was borne out in the results; (6) fine lateral nystagmus. The consistent presence of this phenomenon corroborates to the full

the opinion emphasized in medical literature on the subject, and its value in diagnosis was considered to be second only to the state of the pupils. At the same time attention must be drawn to one significant fact which was revealed in the correspondence (Howells, 1960) that followed publication of my first survey. This was the observation that nystagmus disappeared temporarily when the content of alcohol in the blood reached an exceptionally high level. This was confirmed in this survey in seven cases in which the driver was well and truly drunk and yet nystagmus was not present. In three of the seven cases in which a plea of not guilty was tendered and urinalysis was carried out the blood-alcohol equivalent in each case was over 270 mg./100 ml.

The incidence of the outstandingly significant clinical phenomena in the present series is given in the following table.

TABLE I.—*Outstandingly Significant Clinical Findings*

Agreed to Examination and Certified	Lateral Nystagmus	Widely Dilated Pupils With Little or No Reaction to Light
281	270 (96%)	276 (98.2%)

It is felt strongly that, of the signs discussed, the consistent presence of pupils almost fixed in full dilatation has received insufficient emphasis in the literature.

Two drivers in this series who had been arrested on suspicion of being under the influence of alcohol were proved to be suffering from hypoglycaemia. Each made a speedy and dramatic recovery within a few minutes of appropriate emergency treatment. At first glance both had shown signs associated with a considerable intake of alcohol: grossly slurred speech, staggering gait, disorientation, and so on. Such cases emphasize the fact that one must never take it for granted that alcohol has been consumed nor must one's vigilance in this respect be relaxed for one moment.

Analysis of Figures

Age

The majority of drivers seen (71%) were between the ages of 20 and 40, and most were first offenders who had never before seen the inside of a police station. The bright spot in the analysis was the rarity of offenders in the teens (1%). In an era in which condemnation of youth has been more than fashionable, and even on occasion justifiable, this must be regarded with great satisfaction.

Occupation

In spite of the dangers of disqualification it was found that almost two-thirds of the drivers (63.5%) were dependent on

* Formerly Divisional Police Medical Officer, City of Manchester.