on both occasions. There are numerous reports of recurrences after curettage.

The main controversy concerns aetiology. Jaffe and Lichtenstein\(^1\)\(^\text{--}^4\) both believe that it is a benign tumour of osteoblastic connective tissue derivation. The main arguments against this are the recording of a recurrence after complete excision, the self-limiting nature of the size of the nidus irrespective of its duration, and the intense formation of a perifocal zone of sclerosis.

Lindbom \textit{et al.}\(^7\) described a characteristic angiographic appearance of a small vessel with an irregular lumen supplying a highly vascular area of bone, with an intense circumscribed blush appearing early in the arterial phase and persisting late into the venous phase. The appearance has recently been confirmed\(^8\) and may well be a useful diagnostic test. This finding, together with the vascularity of the nidus, the prominent vascularity of the adjacent soft tissue, and prolonged symptomatology in the absence of definitive radiological changes, suggest a developmental relationship between the nidus and the associated vascular changes. Whether the vascular alterations represent cause or effect is not yet clear.

A vascular origin might also help to explain the intense pain. No abnormal neurogenic tissue or glomus cell hyper trophy has ever been found. Could the pain be due to a large blood supply confined to a small area within rigid walls?

\(^1\) Jaffe, H. L., \textit{Archives of Surgery}, 1935, 31, 709.
\(^7\) Lindbom, A., \textit{et al.}, \textit{Acta Radiologica}, 1960, 54, 327.

\section*{Dangers in Radiology?}

Many professional societies have good records of membership covering long periods, and these are potentially valuable resources for research into the occupational hazards faced by their members. One of the best known examples is within our own profession, where study of the causes of deaths of radiologists has contributed substantially to knowledge of the hazards of radiation. The excess risk of leukaemia in U.S. radiologists had been reported several times before Warren in 1956\(^1\) claimed that radiologists also experienced an additional, non-specific shortening of life, not manifest as any one disease. At about the same time, however, Court Brown and Doll\(^2\) found no excess risk of leukaemia or of general mortality in British radiologists, and Seltser and Sartwell\(^3\) criticized Warren's conclusions on statistical grounds.

The possibility of a non-specific shortening of life has now been raised again. Matanoski, Seltser, Sartwell, and others\(^4\) have looked at mortality in U.S. radiologists up to 1969 and compared their experience with that of their contemporaries who were physicians, ophthalmologists, and otorhinolaryngologists.\(^5\)

Among those joining their respective colleges in 1920-9, and again in those joining in 1930-9, the radiologists had the highest mortality from both cancer and from all other causes. But among those joining in 1940-9 the radiologists, while still having the highest mortality from cancer, had the lowest mortality from all other causes.

Two phenomena are given particular attention in the report. Firstly, the high leukaemia mortality in the first two groups of radiologists was not found in those joining in 1940-9; on the other hand, while the incidence of lymphoma was not increased in radiologists who joined in 1920-9, it was increased in those who joined later. Several possible interpretations, including damage to the immune response, are offered for this. Secondly, why should the early radiologists have had the highest death rates from diabetes, cardiovascular-renal disease, stroke, hypertension, and suicide?

Unfortunately the authors have chosen the wrong denominators for their statistical testing. This is unlikely to have invalidated their findings on mortality from individual diseases, but may mean that the "non-specific shortening of life" is merely variation within limits that may be ascribed to chance. Furthermore, as they themselves say, at least five and perhaps 20 further years' follow-up on the 1940-9 cohort will be needed to confirm some of the trends described. Until this is done, there seems no reason to change our 1958 belief\(^6\) that the case has not been proved.


\section*{Bacteraemia from the Bowel}

The study of transient bacteraemia following therapeutic trauma is founded on two classical examples. That produced by instrumentation in the lower urinary tract was first fully described by Barrington and Wright\(^1\), in whose paper the page heading (though not the title) is "Catheter Fever," the term formerly applied to the clinical manifestation before its nature was known. Five years later Okell and Elliott\(^2\) established the existence of bacteraemia after dental extraction and the factors governing its frequency. Later studies have extended these observations. Indeed the variety of occurrences and manipulations in the mouth said to be capable of releasing bacteria into the blood is surprising. Nevertheless any trauma which exposes severed or torn vessels in a lumen heavily laden with bacteria is liable to have this result, and this means most of the alimentary tract and any infected urinary tract.

By far the most heavily bacteria-laden area of the whole body is the lower bowel. It would be surprising if a few of its myriad inhabitants did not pass through the mucosa. In fact there is evidence that they do in the normal bowel, to be arrested in mesenteric lymph nodes if in the lymphatics or by Kupffer cells in the liver if they enter the portal system. Surgical operation in such an area presents an obvious risk, and septicaemia has been recorded as following biopsy of a rectal polyp\(^3\) and peroral biopsy of the jejunal mucosa.\(^4\) These infections were due to a \textit{Klebsiella} and \textit{Escherichia coli} respectively, the latter in a patient whose jejenum was known to be abnormally colonized by enterobacteria. But several less easily explicable examples of bacteraemia of intestinal origin have recently been reported by Le Frock\(^5\), of West Virginia Uni-
Renal Transplantation in Diabetes

Advanced diabetic neuropathy is a fatal disease. At this stage patients usually have multiple diabetic complications—proliferative retinopathy (many are blind), arterial disease, hypertension, and neuropathy, which may affect the autonomic innervation of the bladder and lead to urinary tract infection. Some of them are too sick for any but the most conservative measures. Chronic dialysis programmes have proved unsatisfactory in comparison with those for non-diabetics. Not only is mortality high, but there is a tendency for neuropathy and perhaps retinopathy to deteriorate. For these reasons many centres are unwilling to include these patients in their dialysis programmes. In contrast, relatively uncomplicated diabetics with glomerulonephritis or other forms of renal disease are usually suitable for long-term treatment and should be assessed more in terms of their renal disease than their diabetes.

A recent symposium in Minneapolis reviewed the management of diabetic end-stage renal disease. The results of renal transplantation were reported from several centres: this is now probably the most promising treatment, especially when live related donors are used. Most experience has been obtained in Minneapolis itself, where of 63 patients there were at the time of the symposium 45 survivors of between 1 and 57 months, 43 of them with functioning kidneys. Survival was, however, less than for non-diabetics, and much poorer results obtained when cadaver kidneys were used. Urological complications after transplantation were commoner in diabetics, and a neurogenic bladder may be a lethal complication because of intractable urinary infection. Major arterial disease also bedevils the diabetic group, and myocardial infarction and strokes were much commoner causes of death than in non-diabetics.

Reversal of the remaining major diabetic complications does not usually occur after renal transplantation. The Minneapolis group believes that visual acuity which had been deteriorating before transplantation tended to stabilize or occasionally improve, though the course of diabetic retinopathy is unpredictable even under more controlled conditions. An improvement of muscle power was observed in patients disabled from motor neuropathy, and this is in keeping with other similar observations in non-diabetics. There was, however, no change in motor nerve conduction. Gastrointestinal symptoms generally improved, suggesting that they were mostly uraemic in origin. There is still no evidence that transplanted kidneys develop diabetic abnormalities; basement membrane thickening has been reported in the longest survivors, but it is not known whether this is a diabetic change or merely a consequence of transplantation.

With the certain knowledge that diabetics with impaired renal function from diabetic nephropathy will deteriorate and die within a short time and with the impression that retinopathy probably advances more rapidly as renal failure advances, it may be argued that transplantation should be performed earlier in diabetics, perhaps when serum creatinine levels are 500-700 µmol/l. Yet the treatment is still hazardous, and despite the considerable advances reported at the Minneapolis symposium the question still needs to be asked on every occasion whether it is justified to admit some of these patients to dialysis or transplant programmes.

---

1 Barrington, F. J. F., and Wright, H. D., Journal of Pathology and Bacteriology, 1930, 33, 871.
3 Lal, D., and Levitan, R., Archives of Internal Medicine, 1972, 130, 127.
7 Le Frock, J. L., et al., Archives of Internal Medicine, 1975, 135, 835.