experiments in particle physics has generated dozens of new devices with at least two radically different solutions on offer for every problem. Almost every experiment contains a large cylindrical chamber to measure the moments of changed particles, but half of them use time projection chambers (TPCs) and the other half jet chambers. A TPC is a completely clear gas volume with a multiwire detector at one end onto which electrons drift in a uniform electric field. It is, in principle, the ideal three-dimensional detector, but the first big TPC had many teething troubles and has only begun to work well in the past two years (D. Nygren, Berkeley, California). Meanwhile the jet chambers, which use shorter drifts and large numbers of wires stretched throughout their volume, have accumulated a very good record of reliability. Their advocates claim that these chambers can resolve bundles (or jets) of closely correlated tracks much better than TPCs. The conference heard detailed reports on new developments in TPCs from Japan and on two detectors for the large electron–position (LEP) collider at CERN, Geneva. There are new jet chambers for the linear electron– positron collider (SLC) at Stanford, California, for the Cornell University e^+/e^- collider (CESR) and for the other two LEP experiments. Each of these jet chambers has different wire geometry and different electronics. It will be fascinating to see who has guessed right.

Perhaps the most ambitious detectors of all are the ring-imaging cerenkov (RICH) counters for the DELPHI expriment at LEP (J. Seguinot, College de France; M. Turala, University of California, Santa Cruz). As in Breskin's detector, the ultraviolet photons from high-energy tracks ionize an organic vapour (TMAE). Electrons from the ionization process then drift for very long distances before reaching the sense-wires. Apart from the mechanical problems of constructing large-area thin mirrors and windows for these counters, there is a fundamental problem in RICH counters arising from the multiplication process. Electron multiplication releases ultraviolet photons that can interact with the TMAE to produce extra spurious electrons. Test results suggest that this problem can be solved by shielding each sense wire from its neighbours with little 'fences' or tubes.

One of the most exciting potential applications of wirechambers is in positron emission tomography. Good spatial resolution is needed for the 511-KeV γ rays from electron–positron annihilation, with a fast data-collection rate to accumulate a picture of the distribution of the positron-emitting isotope inside an immobilized patient. Charpak thinks it can be done with barium fluoride crystals and parallel-plate avalanche chambers. But there are problems as well as several other competing techniques such as the use of bismuth germanate crystals with solid-state photodetectors. There is a good chance that a successful system will be working before the next family reunion in 1989. □

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Immunology

A common form of killing

From Peter J. Lachmann

IT HAS been established for some years that serum complement kills cells by the insertion of pore-forming molecules (plugs) into the target membrane and the suggestion that lymphocytes kill target cells by an analogous process, discussed in a *News and Views* article[1] some time ago, has been amply confirmed[2,3]. Certain bacterial toxins — for example streptolysin-O and staphylococcal alpha-toxins act in a similar way[4] as does the bee venom toxin, melittin, whose pore-forming activity is rather similar to that of complement[5]. The paper by Young *et al.*[6] on page 613 of this issue extends the role of pore-forming molecules to the eosinophil, a cytotoxic cell of the myeloid series which is known to have important cytotoxic activities both in defence against parasitic infections and in the pathogenesis of the cardiac lesions associated with eosinophilia.

The evidence presented by Young *et al.* is based largely upon electrical potential changes in membranes and lipid monolayers, techniques which have been used by other groups to demonstrate pore formation by complement and by this group to confirm the existence of such molecules in the granules from cytotoxic T cells and natural killer cells[7-9] as well as to demonstrate similar pore-forming molecules in *Entamoeba histolytica*[10]. Marker release from liposomes can be demonstrated and molecular sieving is now being used to find the size of the functional lesion. The ultrastructural appearance of the lesions produced by the membrane attack complex of complement, the final pore-forming components, and by perforins, the leukocyte equivalents, has also been extremely helpful in elucidating their nature and it will be of interest to see what the eosinophil pore looks like when seen under the electron microscope.

That the eosinophil can form pores is of particular interest because, like other granulocytes, the eosinophil has another major killing mechanism available to it — the production of oxygen metabolites producing what may be termed 'burns'. Oxygen metabolites formed by the 'superoxide burst' are known to be the principal intraphagosomal killing mechanism for microorganisms[11]. Oxygen metabolites are also implicated in extracellular cytotoxicity brought about by neutrophils and by macrophages[12]. These reactions produce their membrane damage finally by lipid peroxidation and can characteristically be inhibited by catalase or superoxide dismutase or by iron chelators. Because the eosinophil contains a peroxidase in its granules and is capable of producing a superoxide burst, it is likely that it also uses oxygen metabolites for cytotoxicity in some circumstances. It is therefore a clear example of a cell that has, and seems to use, two of the major cytotoxic mechanisms.

The third major cytotoxic mechanism of eukaryotic cells involves the production of soluble cytotoxic factors, of which tumour necrosis factor and lymphotoxin are the best described. It is clear that monocytes use tumour necrosis factor when they kill nucleated cells by antibody-dependent cellular cytotoxicity[13-15] and although the roles of lymphotoxins in killing by T cells (or B cells) remain to be clearly defined, these cells can certainly produce lymphotoxin, which presumably does function *in vivo*.

It is therefore now becoming clear that these three major mechanisms — plugs, burns and poisons — are used in various combinations by different cell types and in different circumstances. Young *et al.* show that eosinophils can use plugs as well as burns. Lymphocytes use plugs as well as poisons and macrophages certainly use burns as well as poisons. It will be interesting to see if any cell uses all three mechanisms. □

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