evidence to suggest intrauterine renal infection has ever been produced. Obstruction due to polypoid and papillary hyperplasia of tubular epithelium may play a part in the initiation of cyst formation both in experimental models of the disease, such as the diphenylamine-induced polycystic disease of rats, and in the human disease.[8] Acquired obstruction of this kind may possibly lead to cystic dilatation of the tubules because of a change in the composition of the tubular basement membrane that causes it to split and renders it less resistant to distension. Adult polycystic disease of the kidneys may, indeed, be due to an abnormality of the genetic locus that determines the structure of the collagen of the glomerular and tubular basement membranes, and the defect in these membranes may be related to a single enzyme deficiency.[4] If that were so identification of the enzyme deficiency might lead to prenatal diagnosis becoming possible.

Meanwhile, the management of adult polycystic disease of the kidneys remains conservative with control of blood pressure and urinary tract infections. Patients should be advised to avoid sports and occupations with a risk of trauma to the kidneys. Drainage of cysts or their decapitation probably has no place; most studies have shown that such surgical interference accelerates the deterioration of the kidney function. A recent report from China[9] claimed that symptomatic improvement was obtained by deroofing the cysts in patients who presented with aching in loins. The Chinese also found that hypertension was more readily controlled after the procedure, but these observations were uncontrolled. Patients with end-stage kidney failure due to adult polycystic disease of the kidneys fare no better or worse than patients with other forms of end-stage kidney disease when treated by dialysis or transplantation. The cysts shrink after such treatment; might this possibly be the result of belated removal of an unidentified cystogenic substance?

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Another look at zinc

A few rare clinical syndromes are associated with low concentrations of zinc in the blood; symptoms disappear when the patient is given zinc.[1] They include a particular type of dwarfism and a specific skin disorder, acrodermatitis enteropathica.

Some biochemical physicians now assert, however, that subclinical zinc depletion may be much more common than clinically obvious depletion.[1-3] Unfortunately, the plasma concentration of zinc is only about 1% of the total body zinc and is not a reliable guide. Another reason, and probably a more important one, for the unreliability of estimations of plasma concentrations of zinc as a clinical pointer is that zinc in plasma is almost entirely bound, largely to plasma proteins, including albumin (50%), α_2-macroglobulin (40%), and transferrin, and to amino-acids (5%). A low plasma concentration of zinc is almost certainly much more commonly due to a change in the concentration of one of these zinc binders than to a true depletion with a fall in the cell content of zinc.

A relation has been shown between the change in plasma concentrations of albumin and of zinc in some diseases[4]—but correction of the zinc for the albumin value would be justified only if albumin was known to be the only zinc binder affected. The presence of several binders probably also explains why there is no predictable relation between plasma concentrations of zinc and protein as they each increase during venous occlusion.[5]

Though large (20%), the fall in plasma zinc after meals is unexplained. It might be due to changes in the concentration of one of the non-albumin zinc binders, though the changes in α_2-macroglobulin are too small to be the explanation. Because of the binding to plasma protein and the effect of meals, plasma concentrations of zinc need to be measured at a standard time and without venous occlusion.

A reduction in concentration of one or more of the zinc binders is the most likely explanation of the fall in plasma zinc by as much as 30% currently being described in so many diseases. These include acute traumatic episodes such as myocardial infarction, chronic "inflammations" such as rheumatoid arthritis, and conditions such as liver disease where there is a change in the plasma concentration of albumin.[4] Indeed, the fall in the plasma zinc concentration after myocardial infarction is so consistent that it has been described as diagnostically useful.[6] This fall has been attributed to an increased uptake of amino-acids (with the zinc bound to them) into the liver under the influence of a leucocyte endogenous mediator[6]—but only 5% of plasma zinc is bound to amino-acids.

The plasma concentration of zinc is reduced by about 20% in patients with rheumatoid arthritis,[7] a change related to the sedimentation rate and to the plasma content of albumin. The fall in albumin could account for the fall in zinc, or possibly some other factor such as the leucocyte endogenous mediator may be at work.[7] Whatever the explanation, the effect on zinc in these various conditions seems to be non-specific and part of the metabolic response to trauma and inflammation.

With zinc (and other trace metals) the general view is that cellular rather than plasma depletion is the important factor that explains symptoms. But what does a low cell content of zinc mean? Most of the zinc in cells is bound to protein or incorporated into enzymes, and any change might be due to a diminished cell content of these zinc binders or enzymes. For example, the low zinc content of erythrocytes in patients with hyperthyroidism is attributed to a diminished cellular content of carbonic anhydrase.[8] Keeling and his colleagues[9] have recently confirmed that in patients with chronic liver disease a low plasma concentration of zinc is associated with, and related to, a low albumin concentration. They showed further, however, that their patients also had a low zinc content in the leucocytes but not in the erythrocytes; some of their patients

may have been zinc deficient, though none had features attributable to zinc depletion. Again, these results might be explained by a diminished amount of zinc binder in the leucocytes.

A cellular effect of a low plasma concentration of zinc has recently been found by Patrick and his colleagues,[10] who showed that the zinc is an important controller of the sodium pump in leucocytes. More recently Patrick *et al*[11] have reported an effect of zinc supplements on sodium transport in children with protein-energy malnutrition. They suggest that there may be a causal relation between the low plasma concentration of zinc and the impaired sodium transport seen, for example, in trauma and malnutrition.

Our knowledge of the chemical anatomy of zinc in health and disease is increasing rapidly. What we know so far suggests that low concentrations of zinc in plasma or cells are not adequate evidence of a primary zinc depletion. The cornerstone of the definition and detection of nutritional deficiencies is a therapeutic test.

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Regular Review

Dysmorphophobia and the search for cosmetic surgery

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The pressures for social conformity and a sense of personal acceptability and worth have always encouraged some people to search for cosmetic solutions. In our own society in the past wigs, bustles, and false busts have been accepted as the norm. Most of our population today wear dentures, at least in part for cosmetic reasons. Nevertheless, those who seek radical and permanent changes in their bodies in an effort to change their appearance, and whose bodies are seen by most others as normal or unremarkable, are somewhat more suspect: the term dysmorphophobic has been coined to describe them.

The anorectic achieves such change in a gross way by pathological slimming, thus banishing the most hated and feared part of herself. The man totally preoccupied with body building seeks an obvious and frequent confirmation of his masculinity. Others turn to surgery to achieve the necessary changes. The feminine need to reduce "fatness" finds expression here in a wish that the surgeon will remove fat from various parts of the body and will reduce the size of the breasts. Breast enhancement, blepharoplasty, and facelifts seem, perhaps, to have a more positive basis with their aim of making the woman feel and appear more desirable, youthful, and adequate. Hair transplants in men may have a similar basis. And what of rhinoplasty? In any one case is it the racial nose or specifically the shape of nose inherited from one or other parent which is now being rejected? Studies have shown that in such circumstances the physical deformities are often minor in themselves; the degree of deformity is unrelated to psychological disturbance—or satisfaction with the effects of surgery.[1 2]

Several studies have shown that, in the short term at least, many people who have persistently sought and undergone such cosmetic surgery have then been satisfied.[3–7] The results are, however, inconsistent: other studies have shown much greater psychiatric morbidity[7 8] and less favourable results of surgery.[9 10] The differences probably reflect differences in the populations studied. Cosmetic surgeons are often well aware of the hazards of their profession. A few patients do badly, becoming obviously and immediately paranoid, litigious, depressed, or suicidal. Such individuals cannot always be easily detected beforehand, when—singlemindedly pursuing their objectives—they may ably conceal aspects of their past and their inner thoughts. Nevertheless, with proper screening some potential morbid psychological responses to surgery can be identified.

Schizophrenics sometimes seek cosmetic surgery in response to or as part of their hallucinatory and delusional experiences and passivity. Schizophrenia is not always a contraindication to surgery,[11] but most often the appropriate medical response will be to treat the psychosis. If schizophrenic symptoms are at all evident then cosmetic surgeons are likely to be wary. If paranoid elements are to the fore then they are certainly wise to be so. Surgery may lead either to further unrelenting complaint about its failure to relieve the deformity, or else the patient may no longer blame his deformity for his problem but