With an endotracheal or tracheostomy tube in place the ventilation may have to be assisted or controlled. Apnoea or severe respiratory insufficiency may be due to permanent damage to the respiratory centre, but it is often difficult to make this diagnosis in the early stages, and controlled ventilation must be instituted until the patient's condition can be more clearly assessed. Another group of patients who may require controlled ventilation are those injured elsewhere in addition to the head; 30% of all head injuries are accompanied by injury to other parts of the body,[1] and, if the associated injury is to the chest, controlled ventilation may be life-saving.[8] The possibility of a chest injury should be considered if, in spite of a clear airway, the patient does not remain well oxygenated.[9] In many patients respiratory insufficiency may be less obvious, and it can then be assessed only by the repeated analysis of samples of arterial blood for oxygen and carbon-dioxide tensions and for acid-base balance. These analyses are also important for the proper control of patients who are being artificially ventilated for long periods. Most hospitals to-day have facilities for measuring carbon-dioxide tension and acid-base balance, but there are still not enough where oxygen tension can be measured. It is to be hoped that all centres dealing with head injuries will soon be able to provide this service. There is still some doubt about the value of humidifying the inspired air for a patient who has been intubated or has had a tracheostomy, but the modern multiple-gauze condenser-humidifier[10] is simple, and since it hardly does any more than take the place of the patient's own upper airway as a humidifier its more widespread use could do little harm and might be beneficial.

Induced hypothermia, with the body temperature lowered to about 33° C. (91° F.), is still sometimes used in the treatment of head injuries, and the anaesthetist, with his specialized knowledge of the procedure, is often called to control the cooling. However, opinion is still divided on the value of induced hypothermia in these cases[5 11]; moreover, it is time-consuming and has some disadvantages and complications of its own.[12] Even those workers who still favour its use accept that hypothermia is of value only if the temperature is lowered within one to two hours after the injury.[11] Here a clear distinction must be made between the artificial reduction of body temperature to 33° C. (the value of which is doubtful in the treatment of head injuries) and the artificial reduction, by the same means, of the body temperature to a normal level in a patient who has hyperpyrexia as a result of injury to the brain stem: the value of this procedure is without doubt. Confusion will continue so long as the word hypothermia is applied to both these entirely different principles of management.[1]

Less than 10% of all patients admitted with head injury require major surgical operation, and many of those with even severe trauma show signs of improvement within a few hours. Many patients who die in hospital after a severe head injury do so as a result of the complications of unconsciousness itself rather than directly from brain damage, intracranial haemorrhage, or sepsis.[5 13 14] It is within these first few hours that the anaesthetist can apply his special skill and knowledge to the care of these patients. His place in the head-injury team is clear; and it is essential, if the respiratory and other difficulties in the period immediately following the injury are to be overcome, that the anaesthetist be consulted early. He can then do much to increase the patient's chances of full recovery. As J. M. Potter[14] has said, "Relatively few head injuries prove to be serious, yet most of us learn sooner or later, that all must be treated seriously if the survival rate is to be maximal."

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[12] Cooper, K. E., and Ross, D. N., *Hypothermia in Surgical Practice*. London. 1960.
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Molecular Biology

To show familiarity with the work of the molecular biologists it is no longer enough for the outsider to remember which glutamic-acid residue has given way to valine in haemoglobin S, or to understand what is meant by a degenerate code. But if the access of new knowledge in the last 10 years has inevitably robbed molecular biology of some of the initial simplicity of its discoveries, this is a price we must gladly pay. In return we have a number of substantial gains. The genetic code has been broken far sooner than seemed probable when its general nature was first appreciated; our knowledge of protein synthesis is gaining rapidly in precision; and from Jacob and Monod's work on the control of gene action, for which they recently gained a Nobel prize in association with Lwoff,[1] have come our first hopes of bringing the study of developmental biology into the molecular fold.

By and large the prophets have proved extraordinarily reliable, even if one allows for the natural tendency of error to be forgotten. From the early confidence of the crystallographers in the 1930s that the biological macro-molecules could be conquered—described in Snow's *The Search*—to the first statement of the type "D.N.A. makes R.N.A. makes protein" the success rate of the predictions made must be the envy of biologists of other kinds. Dr. F. H. C. Crick, F.R.S., in introducing the latest issue of the *British Medical Bulletin*,[2] devoted to molecular biology, is cautious in predicting particular clinical applications of the new knowledge, but he is surely right in implying that we have no real choice but to seek understanding of the processes governing disease at the molecular level.

How we define molecular biology and its relationship to other branches of science matters little. Biochemistry could claim all of it; yet biophysics has provided techniques, genetics and microbiology the material. If these have been the contributors, cell physiology has so far been the major beneficiary. Clinical medicine has yet to be much involved in all this, though the abnormal human haemoglobins have a place in the history of molecular biology, and the crucial discovery of bacterial transformation by D.N.A. was the outcome of a clinically inspired curiosity about the changes bacteria may undergo during infection. This was when F. Griffith[3] showed experimentally that one antigenic type of pneumococcus could be transformed into another.

[1] *Brit. med. J.*, 1965, **2**,
[2] *Brit. med. Bull.*, 1965, **21**, 183–278.
[3] Griffith, F., *J. Hyg. (Lond.)*, 1928, **27**, 113.

For the future there are prospects of control of protein synthesis. To predict early success at a level capable of clinical application would be unwise, though to be entirely pessimistic would be to belie the whole history of this field.

The interference by antibiotics with the synthesis of nucleic acid or protein provides the molecular biologist with a tool, which has enabled him to discover more about their probable mode of action. On the other hand the capacity of populations of pathogenic micro-organisms to become resistant to antibiotics has brought a practical challenge to the molecular geneticist, and one complicated by the discovery that drug resistance can sometimes be transmitted to non-resistant cells by infection. Thus epidemiology, therapeutics, and molecular biology are already in contact with each other.

Molecular biology is sometimes equated with the study of D.N.A., R.N.A., and their role in protein synthesis. It is as well to be reminded that proteins, once synthesized, may have functions in the cell that can be analysed by biophysical, biochemical, and electron-microscopic study. The study of the contractile proteins in muscle cells, discussed in one paper in the *British Medical Bulletin*, is a model of what can be achieved. It seems likely that a similar *Bulletin* published 20 years from now would no longer be dominated by the nucleic acids and protein synthesis. Protein function and the polysaccharides should by then be claiming a larger share of the limelight.

Dermatitis from Lichens

The number of substances known to be capable of inducing allergic sensitization is already vast and receives new additions almost daily through the ingenuity of the industrial chemists. But occasionally a new recruit to the list of offenders is identified in some apparently innocuous and unsuspected object in man's natural environment. Such are the plants known as lichens.

Lichens are very widely distributed outside cities and must be familiar to most people who ever visit the countryside. They consist of a fungus and an alga in symbiosis, and form conspicuous patches of orange, yellow, green, grey, and other colours on roofs, walls, and trees. Forestry workers are most frequently in contact with them, but factory workers, carpenters, gardeners, and housewives may handle lichen-covered logs and branches. Thus casual exposure can occur in many different circumstances. M. L. Spillman[1] in France appears to have been the first to recognize, in 1921, that lichens may cause dermatitis. Occasional further cases of lichen dermatitis have been reported among woodcutters in Switzerland[2] and in France. M. P. Le Coulant and G. Lopes[3 4] thoroughly investigated this problem in forestry workers in the Bordeaux region. The significance of these reports was not generally appreciated, and there can be few dermatologists who seriously consider lichens as a cause of unexplained dermatitis.

Almost simultaneously R. H. Champion[5] in Cambridge and J. C. Mitchell[6 7] in British Columbia recognized further examples of lichen dermatitis. The Canadian patients were both forestry workers, but the English patient was a housewife who had related her dermatitis to contact with wood dust on her husband's clothes. In all these cases patch-tests with lichen were positive, not only with the offending species but with related species in the same genus and other genera in the same order. Mitchell's work has established that the sensitizing agent was usnic acid. The species so far incriminated belong to the genera *Lecanora, Parmelia, Physcia,* and *Xanthoria.* The clinical picture is that of a recurrent acute or chronic dermatitis of the face and neck, hands, and forearms. The dried and powdery lichen may pass the shirt neckband and provoke an extension of the dermatitis around the belt-line and on the genitalia.

Since the species of lichen that have caused dermatitis grow in many parts of the world, these plants must take their place on the list of suspects when patients with contact dermatitis are found to have handled either green or dried wood. It is essential that the diagnosis be confirmed by patch-testing with the lichen itself or with usnic acid. Many people exposed to lichens in Europe will also be in contact with pine, spruce, larch, or common ivy, which may all occasionally cause dermatitis. Rarely the common species of elm may also do so, but the leaves rather than the timber are responsible. A so-called diagnosis based solely on circumstantial evidence is therefore of little value.

Allergic contact dermatitis is an increasingly important hazard of modern life, whether at home or at work.[8] A detailed, skilfully taken clinical history will often allow the large range of possible offending substances to be reduced to a limited number of suspects. These can then be investigated by specific patch-tests, for patch-tests properly carried out and critically interpreted are a helpful procedure in reaching an accurate diagnosis. Success in identifying the offending substance can be achieved, unless by chance, only with a knowledge of the sensitizing potential of each component of the patient's occupational and domestic environment, and this is often very difficult to obtain.

Readmission Rates in Schizophrenia

Patients with schizophrenia are nowadays kept in hospital for a shorter time on the average than they used to be. Treatment outside is proving successful in some cases, and this week Drs. A. Esterson, D. G. Cooper, and R. D. Laing report in the *B.M.J.* (page 1462) their results with 42 young patients, 20 being admitted to hospital for the first time. They were treated by "family and milieu therapy" on discharge from hospital, and only seven of the 42 patients (17%) were readmitted during the year after discharge. The prognosis in a group of this kind would be expected to be better than average, and similar readmission rates have been reported for other series, in which the patients did not have the benefit of such special treatment. Consequently, some confirmation would be welcome of the authors' plea that their results provide "a prima facie case for radical revision" of the treatment of schizophrenic patients. However, one general consequence of the shorter average stay of schizophrenics in hospital is that they are being readmitted after

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[7] —— and Champion, R. H., *Bryologist*, 1965, **68**, 116.
[8] *Brit. med. J.*, 1965, **2**, 1322.