Endemic goiter is widely prevalent in the countries of Southeast Asia; the Himalayan endemic is one of the world's most extensive and severe endemics. It is accompanied in many areas of this region by endemic cretinism, deaf-mutism, and other developmental disorders and thus constitutes a significant health problem. For many years the etiology and pathogenesis of goiter in the Himalayas had remained obscure. Studies of the problem indicate that the endemic in Southeast Asia is primarily the result of environmental iodine deficiency. The thyroid makes a series of finely balanced adjustments to chronic iodine deprivation. We have shown that the Indian endemic can be controlled successfully by the administration of physiological doses of iodine in the form of either potassium iodide or iodate added to domestic salt.

I was trained in the basic science of pathology but in all of the work of me and my colleagues, I commuted freely among laboratory, clinic, and community, with the result that today I am nearly homeless. The study of endemic goiter in the Himalayas had a peculiar fascination that induced me to cut across disciplines.

Endemic goiter in Southeast Asia is an age-old problem. There are references to it in ancient Hindu scriptures dating back to 2000 B.C. “Tumors” of the neck, generally regarded as thyroid swellings, have been described by the Hindu physician Charaka and the surgeon Sushrutha in their treatises from about 600 B.C. The inhabited valleys of the Himalayan massif have long been regarded as one of the world's classic and most intense areas of endemic goiter (1). It was in the study of this endemic that Sir Robert McCarrison labored for nearly 30 years in the early part of this century in search of its causative factors and as a result propounded the theory that the endemic was of complex derivation, being related to "Goitre Noxa" and faulty and deficient diets (2). It was then that excessive intake of calcium through drinking hard water (3) was postulated as the causative factor. Still later, excessive intake of fluoride, because of the high fluoride content of environmental waters, was believed to be the cause (4). By mid-century there was much uncertainty about the cause of Himalayan endemic goiter and very little progress in its prophylaxis. Fortunately, recent developments in the use of radioactive iodine and of physicochemical methods for the study of thyroid physiology led to renewed interest in reevaluating the cause of endemic goiter in many parts of the world, including Southeast Asia, an interest triggered by the pioneering studies of Stanbury and his colleagues in Mendoza, Argentina (5). In several countries of our region the extent of the problem has been redefined, and prophylactic measures are under way (6, 7).

Extent of the Problem

Starting with Afghanistan, in the extreme Northwestern end, goiter is endemic along the northern and southern slopes of the Hindukush mountains, along the banks of the great Oxus river (Figure 1). There are 70,000 persons resident in this zone, and
prevalence rates vary, from 10% to 60%. From the mountain passes of Afghanistan, the goiter belt extends for about 1500 miles into Pakistan and then into India, along the southern slopes of the Himalayas. There are, in addition, isolated pockets of endemicity within the mainland of the Indian subcontinent (6). The population at risk in the vast Indian goiter belt is approximately 40 million, out of which 9 million are believed to be affected with goiter (1, 8). Some of the world’s greatest endemic foci are to be found in the Indian belt, with prevalence rates going up to 90% in the general population. The whole of Nepal is goitrous; prevalence rates range from 7% among males in some areas to 100% among females in some others. There is no disease that affects Nepal so uniformly and to such an extent. The contiguous Himalayan mountain kingdoms of Sikkim and Bhutan are also almost wholly goitrous. In Bhutan, for example, goiter is taken for granted except by some self-conscious young women, and nodular goiter is scarcely regarded as a disease (9). A survey of the Interdepartmental Committee on Nutrition for National Defense, U.S.A., in 1966 found that a fourth of the population of Bangladesh had a visible goiter.

The goiter belt, then, extends from the Naga and Lushai hills in the eastern part of India into Burma, where goiter is widespread. Prevalence varies widely, from about 2% in the lowlands to about 90% in some parts of the Chin Special Division. Several districts of Kachin State have prevalences of from 40% to 60%. In Central and Southern Burma, a survey carried out by the Interdepartmental Committee on Nutrition for National Defense in 1962 recorded prevalences of from 2% to 33%.

From Burma, the endemic extends into the northern and northeastern provinces of Thailand, and, here, several surveys made since 1955 showed prevalences of from 15% to 72%. In Prae province, which was studied extensively before the introduction of iodized salt, prevalence in school children of between 7 and 13 years of age ranged from 10% to over 90% (10). It is estimated that nearly 3.5 million people are at risk in the goitrous regions of Thailand. From the northeastern Thai provinces, the endemic extends into Laos. Goiter has been reported from several parts of Laos, Cambodia, and North and South Viet Nam (6). A survey made by the Interdepartmental Committee on Nutrition for National Defense in South Viet Nam in 1959 found that goiter was not seen in populations living on the Coast, but it was encountered in the Delta and the highland populations, a prevalence of 34% having been recorded in a village in the highlands.

In Malaysia, goiter was reported several years ago in the inland mountains, to an extent of 40% (11), and a recent report describes endemic goiter in a rural community near Pahang (12). A survey made by the Interdepartmental Committee on Nutrition for National Defense in 1962 found that goiter was not an alarming problem in Malaysia, although small pockets of the endemic do exist. Goiter is extremely common in the mountainous areas in the interior of Sarawak along the whole of the border with Indonesia (7). The problem was considered to be serious enough to establish a plant to iodize salt in Sibu in 1959 and another in Kuching in 1967. Endemic goiter was found throughout the length of the Indonesian archipelago (6). Ancient Javanese medical texts suggest that goiter had been endemic in the island of Bali for nearly ten centuries. One of the world’s most severe goiter endemics has been dis-
covered in recent times in both Eastern and Western New Guinea (13-15). Despite the use of iodine prophylaxis in Indonesia for 25 years, a recent report from East Java indicates that goiter is still a public health problem there; in two villages, 25% and 97%, respectively, of the school children showed thyroid enlargement (16).

In Ceylon, goiter occurs in the wet zone in the southwest quarter of the country, which includes the whole of western Sabaragamuwa, the Central and Southern Provinces, and part of Uva province. There are 8.5 million persons in this area who are believed to be exposed to the risk of goiter. Prevalences in school children range from 12% to 54% (7).

Cause

Wherever endemic goiter has been investigated with modern methods in Southeast Asia the findings have been uniform and entirely consistent with the hypothesis that environmental iodine deficiency is the primary cause (13-15, 17-24). There is no definite evidence to incriminate extrinsic goitrogens or intrinsic defects in hormonogenesis, other than a suspicion that buckwheat (Fagopyrum tartaricum) may act as a mild goitrogen in the high mountains of Nepal (22).

Studies by our group in the goiter zones of India, Nepal, and Ceylon have given the following picture (17-21). The iodine content of drinking water in the goitrous localities of India, Nepal, and Ceylon is low in comparison with waters from nonendemic areas and generally less than 3 µg/litre (Table 1). The 24-hour urinary excretion of stable iodide or the urinary excretion of stable iodide per gram of creatinine in a casual urine sample, wherever studied in this region, was generally low in comparison with corresponding values from nonendemic areas (Table 2); the mean excretion levels were less than 50 µg per gram of creatinine, whereas the mean control value in 10 subjects from a nonendemic area was 76.4 ± 10.2 µg/g creatinine. The late Richard Folliis, from a comparative study of school children in Washington (a nonendemic area) and in the endemic areas in Thailand, suggested a urinary excretion of 50 µg of iodide per gram of creatinine as the lower limit of normal (23).

Radioactive-iodine uptakes are elevated, markedly so, even at 4 and 6 hours after oral administration of the isotope; 24-hour uptakes being generally between 60% and 80% of the administered dose (Table 2). Plasma inorganic iodide levels are reduced (Table 3). The serum protein-bound iodine levels are distinctly reduced both in Indian and Nepal endemics, often without evidence of clinical hypothyroidism (Table 3). The reduction in serum protein-bound iodine is not the result of a reduction in serum proteins but has to be regarded as an expression of severe iodine deficiency. A low protein-bound iodine level without hypothyroidism is an intriguing finding, and the explanation may lie in the fact that, in the Himalayas, the circulating thyroid hormone is predominantly in the form of T₃ (triiodothyronine) (22). Protein-bound I₁-thyroid hormone is decreased both in Indian and Nepal endemics, indicating that the gland is under normal pituitary control. These results obtained by our group in India, Nepal, and Ceylon are generally in harmony with those obtained in New Guinea (13-15).

Direct measurement of organic iodine stores in the thyroid at autopsy in India (18) and at biopsy in Thailand (26) showed low concentrations in endemic goiter; 25 to 57 µg per gram of tissue in the former case and 18 to 21 µg/g tissue in the latter, compared

<table>
<thead>
<tr>
<th>Country</th>
<th>Place</th>
<th>Iodine Content of Water µg/litre</th>
</tr>
</thead>
<tbody>
<tr>
<td>India</td>
<td>Uttar</td>
<td>0.298</td>
</tr>
<tr>
<td></td>
<td>Prades</td>
<td>0.205</td>
</tr>
<tr>
<td>Nepal</td>
<td>Bihar</td>
<td>0.125</td>
</tr>
<tr>
<td></td>
<td>Trishuli</td>
<td>0.107</td>
</tr>
<tr>
<td></td>
<td>Jumla</td>
<td>0.25</td>
</tr>
<tr>
<td>Ceylon</td>
<td>Horana</td>
<td>1.25</td>
</tr>
<tr>
<td></td>
<td>Delhi</td>
<td>9.00</td>
</tr>
</tbody>
</table>

Table 1. Iodine Content of Water Samples in Endemic Goiter Areas in India, Nepal, and Ceylon

<table>
<thead>
<tr>
<th>Country</th>
<th>Place</th>
<th>Uptake of I₃¹ in 24 hours %</th>
<th>Urinary Excretion of Iodide µg/g creatinine</th>
</tr>
</thead>
<tbody>
<tr>
<td>India</td>
<td>Uttar</td>
<td>68.7</td>
<td>30.2</td>
</tr>
<tr>
<td></td>
<td>Prades</td>
<td>±8.3 (70)</td>
<td>±2.87 (46)</td>
</tr>
<tr>
<td></td>
<td>Bihar</td>
<td>67.1</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>±12.3 (43)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Nepal</td>
<td>Trishuli</td>
<td>71.6</td>
<td>21.6</td>
</tr>
<tr>
<td></td>
<td>±1.65 (41)</td>
<td>±1.59 (40)</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Jumla</td>
<td>84.7</td>
<td>20.2</td>
</tr>
<tr>
<td></td>
<td>±1.88 (17)</td>
<td>±3.04 (11)</td>
<td>-</td>
</tr>
<tr>
<td>Ceylon</td>
<td>Horana</td>
<td>77.5</td>
<td>20.15</td>
</tr>
<tr>
<td></td>
<td>±1.41 (33)</td>
<td>±3.00 (6)</td>
<td>-</td>
</tr>
<tr>
<td>Control values</td>
<td>Delhi</td>
<td>42.4</td>
<td>76.4</td>
</tr>
<tr>
<td></td>
<td>±3.00 (15)</td>
<td>±10.2 (10)</td>
<td>-</td>
</tr>
</tbody>
</table>

* From Ahuja and Kochupillai (41). Numbers in parentheses indicate number of samples analyzed.
Table 3. Serum Protein-Bound Iodine (\(1^131\)), Protein-Bound \(131^1\)Iodine (\(1^131\)), and Plasma Inorganic Iodide (PII) Values in Endemic Goiter of India, Nepal, and Ceylon:

<table>
<thead>
<tr>
<th>Country</th>
<th>Place</th>
<th>Protein-Bound (1^131)I (\mu g/100 \text{ ml})</th>
<th>Protein-Bound (1^131)I (% \text{ dose/litre})</th>
<th>PII (\mu g/100 \text{ ml})</th>
</tr>
</thead>
<tbody>
<tr>
<td>India</td>
<td>Uttar Pradesh</td>
<td>3.87 ± 0.31 (24)</td>
<td>0.53 ± 0.130 (11)</td>
<td>0.096 ± 0.021 (12)</td>
</tr>
<tr>
<td></td>
<td>Bihar</td>
<td>3.00 ± 0.11 (13)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Nepal</td>
<td>Trishuli</td>
<td>4.10 ± 0.26 (15)</td>
<td>1.87 ± 0.625 (12)</td>
<td>0.088 ± 0.017 (8)</td>
</tr>
<tr>
<td></td>
<td>Jumla</td>
<td>4.43 ± 0.44 (12)</td>
<td>1.53 ± 0.504 (12)</td>
<td>—</td>
</tr>
<tr>
<td>Ceylon</td>
<td>Horana</td>
<td>5.60 ± 1.31 (18)</td>
<td>1.09 ± 0.14 (13)</td>
<td>0.089 ± 0.013 (14)</td>
</tr>
<tr>
<td>Control values</td>
<td>Delhi</td>
<td>6.00 ± 0.70 (15)</td>
<td>0.115 ± 0.02 (15)</td>
<td>0.137 ± 0.018 (10)</td>
</tr>
</tbody>
</table>

* From Ahuja and Kochupillai (41). Numbers in parentheses indicate number of samples analyzed.

with control values that ranged between 246 and 373 \(\mu g\) in the former case and 350 to 700 \(\mu g\) in the latter. Work from Thailand further indicates that where nodular transformation has taken place, especially in older persons, formation of iodothyronines is slow, and there is a high monoiodotyrosine-to-diiodotyrosine ratio, with iodothyronines predominating in the gland (26). There is evidence of continued and intense hyperplasia of the thyroid in the Himalayas from birth onward, with little or no opportunity for involution and colloid accumulation throughout the lifetime of the thyroid (27). This is regarded as an expression of the severity of the endemic.

There is thus overwhelming evidence that iodine deficiency is the primary etiological factor in endemic goiter in Southeast Asia; the responses of the thyroid can only be explained by iodine deficiency.

Studies in Goats in the Himalayas

Further evidence for environmental iodine deficiency as a major factor has come from studies made on the thyroids of goats living in the goiter belt in India (28). As in humans, the goat thyroids are enlarged even at birth and are markedly hyperplastic, with little or no colloid (Figure 2). Their organic iodine content, whether expressed per unit weight of the gland or per total stores in the gland, is greatly diminished in comparison with that of goats of similar body weight from a nonendemic area (27). Their radioactive-iodine uptakes are elevated. Enzymic hydrolysis and chromatography of homogenates of thyroids labeled in vivo with \(131^1\)I 48 hours earlier showed interesting changes in the distribution of iodoamino acids (28). The percent distribution of radioactivity in the iodoamino acids in the goitrous goats showed higher monoiodotyrosine-to-diiodotyrosine and triiodothyronine-to-tetraiodothyronine (thyroxine) ratios than those from the control areas. This pattern is consistent with iodine deficiency in man and in experimental animals. With the relative reduction in iodine-rich compounds such as \(T_4\) (thyroxine) and diiodotyrosine, linked to a decrease in the concentration of iodine in the gland, it is possible that there is a farther spacing of diiodotyrosine residues in the thyroglobulin molecules, without enough iodine to permit coexistence of the two diiodotyrosine residues.

Polyacrylamide-gel electrophoresis of iodoproteins of goat thyroids labeled 48 hours earlier with \(131^1\)I in vivo showed a relatively higher incorporation of \(131^1\)I into 27S iodoprotein and correspondingly less in

Figure 2. Characteristic histological features of intense hyperplasia in the thyroids of goats in the Himalayas. (Hematoxylin and eosin; magnification, \(\times 140\).)
the 19S moiety, compared with control nonendemic goat thyroids (28). Presumably, there is increased biosynthesis of 27S as a transition from 19S in iodine deficiency, but this has yet to be demonstrated directly. What functional benefit accrues to the animal by this shift in distribution of iodoproteins of thyroid is not clear. Nevertheless, it is clear that the gland preferentially synthesizes T$_3$ to T$_4$.

Here then is a series of finely balanced reactions of the thyroid in man and animals in the Himalayas—enhanced trapping of iodide; increased cell mass, with decreased follicular size; taller and bigger lining cells; maximal organification of iodine; preferential formation of T$_3$ and its secretion into the circulation, rapid release of iodothyronines; greater iodination of 27S iodoprotein, presumably reflecting enhanced formation of this fraction—all of which represent the spectrum of thyroidal response to chronic iodine deprivation. It is difficult to visualize a more complete use of available iodine in Cannon’s “wisdom of the body.”

Natural History and Health Consequences

Endemic goiter is an expression of the biological adaptation of the thyroid to insufficiency of iodine. In areas of mild endemicity it manifests selectively in groups exposed to physiological stresses, such as school-going children around puberty and pregnant and lactating women; female-to-male ratios of prevalence are high. There may be seasonal fluctuations in incidence because of varying iodine supplies (29). The greater the severity of the endemic, which in this region is nearly tantamount to the degree of iodine deficiency, the younger the age groups affected—so much so that in superendemic areas such as the Himalayas, the gland is enlarged to two or three times normal, even at birth. The transformation from simple diffuse enlargement of the gland to marked enlargement with nodular change occurs more readily in younger age groups in those areas where the endemic is more severe; male-to-female ratios tend to equalize (30).

Apart from the disfigurement caused by visible goiters, big goiters, which are often multinodular, frequently undergo hemorrhagic and cystic change in the nodules and cause respiratory distress that requires surgery. Such changes are common in the countries of this region, and the demand for surgery is great.

In the highly goitrous regions of the Himalayas in India and Nepal and in Burma and Thailand, one sees an appreciable number of persons with varying degrees of developmental disturbance: “feeble-mindedness,” cretinism, deafmutism, and other developmental disorders (Figure 3). McCarrison, in 1908, distinguished between “nervous” cretins and myxedematous cretins (31). The general impression has been that where the goiter rate in a population rises above 50% these tragic sequels begin to appear in the population (7). In the Gilgit area where McCarrison made his original observations, recent studies indicate that 2.4% of the population were deaf-mutes (32). In some of the affected villages in the Himalayan tracts in Uttar Pradesh in India, Stott, in 1931, estimated that 4% were deaf-mutes (3). In another area to the west of these foothills 1% of the population showed mild or extreme forms of mental and physical retardation (33). In the northern Himalayan valleys of Nepal, where goiter is superendemic, a high frequency of these malformations is observed. In the Jumla valley, for example, where almost everyone has a goiter, 8% of the population is estimated to be affected with these malformations (20). In the Khumbu region to the northeast, on the southern flank of Mount Everest, at an altitude of 13 000 to 16 000 feet, 98% of the population have goiter, 5.7% are cretins, 12.1% are deaf-mutes, and nearly a third of the population show clinical features of hypothyroidism (22). Among the pigmy tribes called

Figure 3. An endemic deaf-mute with mental deficiency.
Tarons in the Adung Long river valley in the northernmost part of Burma, the Burma Medical Research Society found a high prevalence of goiter but only a few cases of cretinism (34). High prevalences of mental defects and deaf-mutes have been reported in some goitrous villages of Burma and Thailand, and in a Burmese village 17.8% of the population were in this category (7). Widespread prevalence of endemic cretinism, predominantly characterized by multiple neurological deficits, has been reported from Eastern and Western New Guinea, and a symposium was held recently in New Guinea on endemic cretinism (35). Fragmentary as these evidences are, they nevertheless point to the magnitude of the serious developmental disorders that accompany severe endemic goiter in Southeast Asia. An accurate clinical characterization of these disorders and definition of their pathological features are urgently needed. This is an uncharted sea at the present time (35).

These deformed men and women do not show any specific disorders of the thyroid, in handling iodine and making thyroid hormones, that would distinguish them from the others, with or without thyroid enlargement, who are living in the same environment. Their radiiodine uptakes, serum protein-bound iodine levels, protein-bound $^{131}$I, and urinary-iodide excretion patterns are similar to those with endemic goiter but without these developmental disorders. The relation of these disorders to iodine deficiency and thyroid-hormone deficiency is not clear, but they are known to disappear with iodide prophylaxis in other parts of the world, and the recent report that a reduction in the prevalence of endemic cretinism in New Guinea, after a program of injection of iodized oil (given before conception), has occurred (36) lends further support to the idea that iodine may play a critical role in a certain stage of development of the fetus (19).

Secondary thyrotoxicosis and thyroid carcinoma do not seem to have a predilection to occur more frequently in endemic than in nonendemic areas in countries of this region (37).

Control Measures

Until recently, the objective of goiter control operations, regardless of the existence of goitrogenic factors, was to ensure a continuous supply of iodine in physiological amounts. Although water, bread, and tablets have been used as vehicles for carrying iodine, fortification of common salt with potassium iodide or iodate is clearly the most feasible method. This is the method being used in several countries of this region. A method that is being tested in different parts of the world in recent years is injection of iodized oil at intervals of a few years. This method was originally introduced in New Guinea, in 1957, by the Australian investigator McCullagh for the control of goiter, and it has given striking results (38). Although it has many advantages, factors such as the lack of manpower in the health services and adverse reactions need to be taken into account before adopting this method on a national scale.

The salt habitually consumed in this region is derived from the sea or rock; it is generally moist, coarse, and impure. The use of iodate in place of iodide and the recent technological developments that enable the use of practical methods of processing crystal aggregates and spraying iodate solution on salt crystals of varying sizes have now made it possible to iodate effectively tropical salt of this region.

Governments in this region have initiated programs for the control of endemic goiter that are in different stages of planning and implementation. The level of iodisation must depend on local factors, the intensity of the endemic, and the presence of goitrogenic factors. As a general rule, it would be preferable to start with relatively higher levels to make a substantial impact on goiter prevalence in a short period of time and to replete the iodine stores of the population. Later, in the “maintenance phase,” the level of iodisation could be lowered.

In India an investigation was made to test the effectiveness of potassium iodide and potassium iodate in the control of Himalayan endemic goiter when these compounds were added in physiological doses to domestic salt in the Kangra valley (33). In a prospective study lasting 5 years and involving a population of nearly 100 000, a striking reduction in the prevalence of goiter was observed in those areas receiving the fortified salt. During the same period the prevalence of goiter remained unchanged in the control zone, which received plain, unfortified salt. In children of school age, goiter prevalence decreased from 37.6% to 19.1% in the zone receiving iodized salt and from 38.4% to 14.6% in the zone receiving iodated salt; in the control zone receiving plain salt, the prevalence showed a slight but not significant rise, from 37.8% to 40.3%. A recent resurvey of this area in 1968 showed a further drop in the prevalence of goiter in the zones receiving fortified salt, to almost negligible levels in the population. This drop was accompanied by a reversion of the iodine kinetics in the thyroid to within the normal pattern (39). Gratifying responses have also been reported from Thailand, indicating a decrease in the prevalence of goiter after iodation of salt. There can be no doubt that endemic goiter can be readily controlled, as has already been demonstrated in the countries of this region.
region, by iodation of common salt. This is a simple, inexpensive, and effective method. Many countries of this region have already embarked upon national goiter programs that use this method.

Conclusion

Endemic goiter is an ancient affliction of mankind. Iodine deficiency has long been regarded as a major causative factor. As newer and more precise methods of study became available this hypothesis has gained further ground. The developments in Southeast Asia in this field followed a similar pattern. We have come a full circle, a little wiser perhaps, back to the position of Chatin, the French chemist who in 1850 claimed iodine deficiency to be the cause of goiter and cretinism, and of David Marine, who in 1908 advanced the hypothesis that endemic goiter resulted from a deficiency of iodine (40). There are now “new clothes over an old body.”

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Confidentiality of Medical Records

The problems of the confidentiality of medical records and of access to them may be divided into two categories. The first relates to access in connection with the treatment of the patient concerned. Witts has pointed out the widespread effects on records procedures of the increasing complexity of medical care. A relationship which, 50 years ago, was usually between one doctor and his patient in the privacy of the home, now involves a team, not only of doctors with different skills, but also of nurses, secretaries, technicians, medical-records librarians, radiographers and laboratory workers. It is in the patient's interest that these people should be informed about the nature of the illness if they are to be in a position to give effective professional assistance. Absolute privacy would mean that the patient could depend for help on the limited skills of one brain and one pair of hands alone. As Witts says, "the confidential relationship is now between the patient and the hospital as a whole and it is not confined to a single doctor". A more difficult issue is raised when data about a particular patient are needed in connection not with his own treatment, but for research purposes. In such circumstances, whose permission is necessary before information about a patient may be released? In some parts of the world, there is a body of opinion which would preclude the communication of medical data for research purposes to anyone, not excluding doctors, without the patient's written consent. Wagner regards the West German code of professional ethics, expressing the principle of secrecy in absolute terms and allowing no exceptions, as the obstacle which makes epidemiological research almost impossible in the Federal Republic of Germany. In other areas, there is a long-established tradition that medical men have discretion to pass on information to other medically qualified colleagues engaged in research without the prior consent of the patient, provided they are satisfied that the patient will not suffer, and provided the research seems in the general interest. A rational solution of this problem must be based upon a realistic evaluation of the importance or otherwise of medical records research. If one agrees with Lord Platt's viewpoint that "occasionally matters of great importance will come from them but more often there is little to be learnt which is not already known", it clearly does not matter whether medical records are made available for research or not. On the other hand, if medical information about identifiable men, women and children is seen as a vital resource in the pursuit of health, the situation is different. My own view is that the latter assessment is correct, and that in the future, as new industrial processes and new fuels create new hazards to health, and more powerful prophylactic and therapeutic agents with unknown long-term actions are brought into being, the public importance of these private records will increase further.

E. D. Acheson
The patient, his record and society.