# Papers and Originals

## Facial Paralysis*

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At first sight facial paralysis seems a banal and even uninteresting subject. After all it is a common condition, often of no great clinical importance, and we deal with most of our cases by rule of thumb. In fact, it poses a number of intriguing and unanswered questions in the fields of anatomy, neurophysiology, and pathology. Its systematic consideration covers a wide range of general medicine, and it affords a promising field for research by the subtle techniques of the applied neurophysiologist, as well as by the simpler methods of the clinician.

If there is one particular lesson to be learnt from a review of this subject it concerns the extent to which therapeutic practice depends on personal impression and uncontrolled observation, rather than on any scientific or rational basis. Nowhere is this more evident than in the case of Bell's palsy, which is bound to occupy a good deal of any discussion devoted to the problem of facial paralysis.

An extensive review of the world literature has yielded no single shred of scientifically valid evidence that surgical treatment exerts a favourable influence on the course of this disorder, yet there are surgeons all over the world who profess an almost religious conviction of its efficacy, and who apparently perform an operation of one kind or another on practically every case referred to them. Some such enthusiasm may of course be a spontaneous manifestation of that innate physical energy and *joie de vivre* that so often distinguishes the surgeon from his more debilitated medical colleagues. On the other hand, I do not find it easy to dissociate the repeated and urgent pleas that reverberate through the American literature describing Bell's palsy as a surgical emergency and begging the physician to refer it at once to the nearest otolaryngologist from the special economic conditions of practice in the United States.

I am reminded of a revealing comment of that great medical historian Henry Sigerist, of Johns Hopkins University, 30 years ago. He observed that the treatment of chronic rheumatism in the United States comprised a prolonged series of expensive injections carried out in the physician's office; in the Soviet Union it consisted of a protracted and expensive course of physiotherapy carried out in a trade union nursing-home on the shores of the Black Sea. We have no evidence to show which of these was the more effective routine, or indeed whether either procedure influenced the course of the patient's disease for better or for worse. What this contrast certainly did reveal was the potent influence of social and economic considerations on medical practice in general and our therapeutic habits in particular. Perhaps the slight hint of therapeutic nihilism that our Continental visitors detect in the British approach to the treatment of facial palsy may not be entirely unconnected with the fact that we work in a professional environment where it is necessary neither to ensure the patient's gratitude nor to impress him with the dynamism of our therapeutic approach.

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### Clinico-anatomical Considerations

The diagrammatic anatomy of the medical neurologist is of course a far cry from the flesh-and-blood anatomy of the surgeon. It is true also that there are many aspects of the finer structure and function of the facial nerve about which we remain woefully ignorant. However, we know enough to allow us to localize anatomical lesions of this particular nerve on the basis of simple clinical examination, an exercise always pleasurable to the neurologist. Here we will first discuss some of the commoner types of facial palsy as they arise from lesions at various sites on the motor pathway.

### Supranuclear Lesions

In the first place it must always be remembered that facial paralysis or weakness may be due to a lesion above the nucleus of the nerve, affecting the corticopontine tract at any part of its course. It may present as an isolated facial paralysis due to a cortical or subcortical lesion, or alternatively as part of a hemiparesis in which infarction or tumour has involved the corticopontine and corticospinal fibres where they are more closely packed together in the neighbourhood of the internal capsule, or lower still in the brain stem. The facial paralysis of a corticopontine tract lesion is characterized by sparing of the upper face. It is not infrequent for a neurologist to be called out to see a patient who is believed to have had a stroke and to observe at once the paralysis of the whole face on the one side that enables him to dispense reassurance that there has been no serious cerebral accident and that the patient is suffering from nothing more serious than a simple attack of Bell's palsy. The upper face escapes in corticospinal tract lesions, because the neurones of the facial nucleus that control movement of the upper face receive impulses from both cerebral hemispheres, while the neurones of the facial nucleus that control the movements of the lower face are activated only by impulses reaching them from the opposite hemisphere.

There is another intriguing feature of facial weakness caused by lesions of the upper motor neurone. Sometimes the involuntary movement of the face that occurs on smiling is retained and may be more than usually conspicuous, even though the patient cannot contract the same muscles at the side of the mouth at all on voluntary movement. This may be very striking, and it is of course never encountered in lesions of the lower motor neurone, where the final common pathway is interrupted and the whole face is paralysed for movement of any kind. There is also a less frequent condition in which the converse is observed: voluntary movement of the face is normal, but emotional and involuntary movement paralysed or weak. This also arises from lesions above the nucleus, but not from damage to the corticopontine fibres of the pyramidal tract. The sites of the lesion or lesions that produce this so-called mimetic facial paralysis are uncertain. They are usually at or below the thalamic level. Sometimes at least

they are in the corpus striatum and may be regarded as a localized variant of the remarkable mask-like bilateral facial weakness that is seen in advanced parkinsonism.

There is one other feature of supranuclear palsy that deserves mention. For reasons that are obscure, disease in the temporal lobe sometimes produces facial paralysis closely simulating that caused by a lesion of the opposite facial nerve.

## Nuclear and Infranuclear Lesions in the Pons

In the pons, facial weakness may be caused by damage to the nucleus of the nerve, or to its fibres as they pass through the pons in close relation to the sixth nerve nucleus to emerge from its lateral aspect. So there are really two anatomical varieties of intrapontine facial paralysis—nuclear and infranuclear. The widespread employment of vaccination against poliomyelitis may shortly render this disease obsolete, but there can be no doubt that typical lower motor neurone facial paralysis may be a sign, and indeed the only neurological sign, of an acute attack of paralytic poliomyelitis. We are not very likely to make a correct diagnosis of poliomyelitic facial paralysis except under epidemic conditions. On the other hand, such cases have occurred in every major outbreak. There is one clinical feature that might lead the observer to suspect a poliomyelitic aetiology. Poliomyelitic paralysis does not develop out of the blue in a perfectly fit patient. No matter how limited the paralytic phenomena there is likely to be a recent history of fever, headache, and neck stiffness that is lacking in the ordinary case of Bell's palsy. A clue in this connexion may also be furnished by the presence of fasciculation in the facial muscles. This may occur in any severe acute lower motor neurone lesion, but it tends to be more prominent and more constant where the nucleus itself is involved, and should lead one to suspect a nuclear rather than an infranuclear lesion.

If we seek an example of facial palsy of peripheral type arising within the pons from the emerging fibres rather than the nucleus of the seventh nerve, we need look no further than multiple sclerosis. It has often been remarked that facial paralysis is the only lower motor neurone lesion encountered in this puzzling disorder. It is not uncommon, and may either herald the onset of the disease or occur at any time during its course. Like most of the lesions of multiple sclerosis, facial palsy tends to recover. It can sometimes be distinguished from Bell's palsy by the absence of initial pain and by the fact that taste is not involved. This is, of course, because the lesion is due to a patch of inflammatory oedema or demyelination within the white matter of the brain stem, where the afferent fibres conveying taste in the accompanying *nervus intermedius* of Wrisberg have already left the entering facial nerve trunk on their way to the medullary *nucleus solitarius*. Unexpectedly, the facial paralysis that develops in the course of multiple sclerosis is infrequently associated with the concurrent involvement of the sixth nerve that might well be expected in view of the site of the lesion, or with the severe and prolonged vertigo that characterizes a more diffuse acute pontine lesion in multiple sclerosis.

The fact that flaccid palsy of the face may originate in the brain stem and not merely from the banal lesion of the facial nerve associated with the name of Sir Charles Bell emphasizes the need for neurological examination in facial paralysis to ensure that more generalized or serious disease of the nervous system is not overlooked.

## Facial Nerve in the Cerebellopontine Angle

When the seventh nerve leaves the lateral surface of the pons it has a short intracranial course in the cerebellopontine angle before it enters the internal auditory canal, and in this situation it is susceptible to a number of pathological processes that often involve paralysis of other cranial nerves. The commonest cause of unilateral facial palsy originating in this part of the nerve is a space-occupying lesion, characteristically acoustic neurofibroma. Meningioma, chordoma, and aneurysm of the basilar artery are less frequent. Curiously, taste is often spared in lesions here. Paralysis arising at this site is often bilateral and may occur in any variety of chronic meningitis. The successes of chemotherapy have reduced the number of such cases, and today even syphilitic basal meningitis is a rarity. At the present time the commonest form of chronic meningitis is carcinomatosis, which often affects the facial among other cranial nerves.

Involvement of the nerve in its intracranial course before it enters the auditory canal is also the source of the bilateral facial weakness often encountered in the Guillain–Barré syndrome, a widespread lesion, certainly inflammatory and probably allergic, characterized by demyelination of proximal nerve roots with relative preservation of the axonal fibres themselves.

## Lesions in the Internal Auditory and Facial Canals

Most cases of facial palsy originate in the bony canal that the nerve traverses between the internal auditory meatus and its emergence at the stylomastoid foramen. The most important cause within the internal auditory canal is acoustic neuroma, but whatever the lesion anatomical considerations predicate the frequent involvement of the eighth nerve. In severe cases paralysis of the greater superficial petrosal nerve, which leaves the geniculate ganglion, causes loss of both affective and reflex tear formation, and interruption of conduction in the chorda tympani leads to loss of taste in the anterior two-thirds of the tongue with diminished salivation. Where the patient escapes deafness, paralysis of the nerve to the stapedius occasionally leads to hyperacusis. Rarely a neurofibroma originates from the sheath of the facial nerve itself with similar results, and in exceptional cases changes seen on an *x*-ray film may indicate the origin of a similar syndrome in an intratemporal dermoid cyst.

The geniculate ganglion owes its picturesque reputation in clinical neurology to the historic contribution of Ramsay Hunt in 1907, who described the condition known as geniculate herpes—a syndrome consisting of acute facial paralysis and a rash in or behind the ear, with or without deafness and vertigo. That such a syndrome is occasionally encountered cannot be denied, but that it arises from changes in the geniculate ganglion itself is open to question. The frequent relation of the syndrome to herpes zoster is indubitable, and has been confirmed by serological tests. However, the situation of the rash is inconstant, and it may be present on the tongue, in the trigeminal distribution, or even in the upper cervical region. Zoster is a general infection by a neurotropic virus, and the fact that every level of the sensory system is pathologically affected probably accounts for the extreme intractability of post-herpetic neuralgia. Facial palsy is one of the uncommon motor complications of zoster which are probably due to a spread of virus from sensory to motor neurones, producing a lesion comparable with that of poliomyelitis, rather than to the inflammatory swelling of the nerve trunk in the facial canal so often suggested as its basis. If the paralysis is indeed due to invasion of the motor nerve cell by virus, peripheral decompression of the nerve is unlikely to help.

There are some conditions other than Bell's palsy, chronic otitis, cholesteatoma, and mastoidectomy that may affect the facial nerve in its bony canal. Among the most important is head injury, and this presents some points of special interest. The first is that post-traumatic facial paralysis nearly always recovers spontaneously. I know that some surgeons operate

on these cases as a routine ; their example is not followed by English neurosurgeons. Indeed, my own experience and that of the Newcastle School of Neurosurgery, as well as that of such authors as Ritchie Russell (1960) and Brodie Hughes (1964), indicate such a good outlook as to permit the argument that leaving the condition alone produces results that are actually better than, and not merely as good as, those of operation. The small proportion of cases where there is no recovery arise where the facial nerve has been actually torn across in a severe fracture of the middle fossa. With this exception post-traumatic facial palsy nearly always shows good recovery, whether it is sustained at the time of injury, develops within two or three days, or—as occasionally happens—begins two or three weeks after the accident.

Facial paralysis complicating head injury is, of course, commonest where there has been bleeding or a discharge of spinal fluid from the ear, and there is often coincident deafness from middle-ear damage. Taste is frequently involved.

There are some rarer but interesting causes of paralysis of the facial nerve arising in this part of its course. It may be compressed by leukaemic deposits. It is a well-known occasional manifestation of severe hypertension, occurring acutely, for example, in the child with acute glomerulonephritis or in the pregnant woman with toxaemia, probably due to haemorrhage into the canal. Sarcoidosis is another occasional cause in a young patient, with a negative Mantoux reaction and often with uveoparotid fever or systemic evidence of sarcoidosis. In most such instances the paralysis is bilateral and is due to granulomatous involvement of the nerve either within or just outside the auditory canal. Again, although decompression has been advised, spontaneous recovery is usual.

Before we consider Bell's palsy, some lesions of the facial nerve after it has left the stylomastoid foramen deserve brief mention. The trauma of a stab wound or of parotid gland surgery are occasional causes. Prolonged excessive opening of the mouth during dental procedures is occasionally responsible. However, probably the commonest mechanical cause is forceps delivery or difficult labour, and in these instances the complete or partial flaccid weakness of the face that ensues usually recovers within a week of the baby's birth. Parotid tumours themselves often cause facial paralysis, and this tends to be subacute and to involve the branches of the nerve unevenly and progressively. Leprosy produces a rather similar neurological lesion, but it is worth noting that this is the only cause of bilateral facial paralysis in which the upper face is more often affected than the lower.

## Bell's Palsy

Bell's palsy can often be distinguished from other forms of facial paralysis by the local pain that precedes it, usually with tenderness on pressure over the stylomastoid foramen in front of the ear, and also by the acuteness with which the paralysis develops. Often this is already virtually complete when the patient becomes aware of its existence, and it is exceptional for its development to occupy more than a few hours. Food sticks uncomfortably in the cheek because of buccinator paralysis, inadequate closure causes the eye to water, and in the elderly all the muscles of the affected side of the face sag. Slight deafness and tinnitus may occur but are always transient. The ear-drum may be pink and congested. Loss of taste occurs in about 10% of cases: for the most part these are more than averagely severe, and the symptom is of unfavourable prognostic significance. Though the sensory fibres of the facial nerve are probably involved in severe cases, the loss of sensation behind the ear that might be anticipated is never encountered, almost certainly because of overlap.

The history of an acute and non-progressive lesion is characteristic, and though this may also be a feature of facial paralysis in multiple sclerosis (unilaterally) and the Guillain–Barré syndrome (bilaterally) pain is absent in both these instances.

Differential diagnosis is implicit in the accounts that have already been given of other kinds of facial paralysis. Under outpatient conditions middle-ear disease, significant deafness, hypertension, involvement of other cranial nerves, and evidence of organic disease of the nervous system must all be carefully excluded. If these precautions are taken in cases where the history is typical, the chances of serious diagnostic error are probably less than in most neurological disorders. The serological exclusion of syphilis is traditional, but I have never found it contributory, nor have I ever found facial paralysis to be a harbinger of diabetes. It is more important to bear in mind that *progressive* paralysis virtually excludes the banal diagnosis.

Recurrent Bell's palsy rightly arouses suspicion, but the condition is not very uncommon, especially in younger patients, in whom the nerve may be affected on both sides at different times. The occasional familial incidence of this syndrome suggests the incrimination of some minor anatomical anomaly.

## Prognosis and Sequelae

Arguments about prognosis are confused by a frequent failure to distinguish complete from functionally adequate recovery. It is certainly true that nine out of 10 patients with Bell's palsy do well enough from the functional and cosmetic points of view to make routine surgery unjustifiable. This is not to say that they recover completely. In some, either inappropriate associated movement or a shadow of residual weakness is detectable on careful examination, but amounts to nothing in the way of actual disablement. If any voluntary movement whatever is present five days after the onset, functional recovery will be nearly perfect, even where some evidence of denervation and reinnervation remains.

The fact remains that in about 10% of all cases there is persisting paralysis, often with troublesome permanent sequelae. Nearly all these patients have shown a complete loss of nerve conduction within a few days of onset, and in most of them taste has been noticeably impaired. The sequelae include ectropion, facial contracture on the affected side, inappropriate contraction of the affected side of the face on contraction of the normal side, and occasionally clonic facial spasm or crocodile tears, both of which usually begin some months after onset. Numerous explanations have been offered to account for crocodile tears, but the likeliest is misdirection to the lacrimal of regenerating fibres intended for the salivary glands. The failure of co-ordinated facial expression evident in closure of the eyelid on opening the mouth after recovery from Bell's palsy is also probably due to misdirection of regenerating axones.

## Early Recognition of Unfavourable Cases

The real issues concern, firstly, the early recognition of this small proportion of unfavourable cases, and, secondly, a critical examination of the evidence that any of the surgical measures advised have been convincingly shown to improve the outcome.

In about 50% of all cases conduction time in the peripheral part of the facial nerve is never impaired, electromyographic fibrillation never appears, and recovery will be complete. In rather more than 30% a transient slowing of conduction indicating partial denervation occurs, but functional recovery is complete. In a minority, however, the nerve becomes unresponsive to stimulation within a few days of the onset and denervation is complete. It is this group that furnishes the residue of cases with persisting paralysis and the com-

plications already described. Electromyographic evidence of fibrillation may be delayed for two to three weeks, and is therefore of little value in any early decision about surgical decompression. Since perfect or at any rate adequate recovery is almost certain unless complete denervation is present at the end of the first week, conduction velocity should be measured at this stage in patients who show no sign of recovery. The high correlation of denervation with loss of taste confirms the prognostic value of this symptom.

Dr. Taverner has been a pioneer in this field (Taverner, 1959 ; Langworth and Taverner, 1963). It is clear that the combination of simple clinical and electrophysiological data facilitates prognosis and the formulation of criteria on which to base controlled therapeutic trials.

Unfortunately the proponents of surgical decompression can quote no such controlled observations, and base their practice on the uncertain grounds of theoretical justification and clinical impression. To the best of my knowledge the nearest published approach to a controlled trial of this form of treatment is found in some fragmentary but suggestive observations made at the Royal Free Hospital in London (Groves, 1965). Groves selected 10 cases of Bell's palsy in which nerve conduction had been lost, thereby fulfilling Dr. Taverner's criteria for complete denervation, and decompressed the nerve in five of these patients shortly after onset. These five did worse than a similar group who declined operation (Groves, 1967). The figures are small, but the value of decompression would need to be firmly established on the basis of similarly strict criteria.

## Where is the Lesion ?

The site of the lesion in Bell's palsy has been sought by neurophysiological methods and by surgical exploration. Jepsen (1965) and Zilstorff-Pedersen (1965) used similar techniques, comprising quantitative studies of taste, elicitation of the stapedius reflex, and measurement of lacrimation. Their results were identical. Except in a very small minority of cases the lesion was localized below the geniculate ganglion, and in about half below the origin of the chorda tympani. Of those above the chorda tympani about half are above the nerve to the stapedius and half below. In other words, the findings of applied neurophysiology support those of the many surgeons who have decompressed the nerve, and confirm Sir William Gowers's clinical pronouncement that the lesion involves especially the part of the nerve trunk that lies within the lower part of the facial canal.

Recalling similarly graphic descriptions of the operative appearances of the sciatic nerve in syndromes which we now know to have had their origin at a much higher level from disc prolapse, the physician may be pardoned an unworthy suspicion that the surgeon who describes the swollen and congested facial nerve observed at operation for decompression is less likely to be familiar with the appearance of the entirely normal nerve under similar circumstances. He may even have some reservations about statements that the histopathological findings encountered in the very rare cases that have come to necropsy favour an ischaemic rather than an inflammatory pathogenesis, or that swelling of the nerve within its sheath is more significant than compression by the bony canal. However, he will be more impressed by experimental observations that transient facial paralysis can be produced in the experimental animal by injection of saline into the nerve within but not outside the facial canal (Jain and Sharma, 1964). This experiment may well be relevant to both the pathogenesis and the prognosis of Bell's palsy. It would fit well with the varying severity of the lesion and with the concept of an initial simple conduction block, below which the nerve will remain healthy, with a normal conduction velocity, unless compression is sufficiently severe and prolonged to cause death of the axone below. Such a view of the lesion is compatible with everything we know about the pathogenesis of Bell's palsy, but contributes nothing to aetiological knowledge, a situation with which we are all too familiar in neurology.

## Aetiology

Both sides of the face are equally affected, the disease is somewhat more frequent in older age groups, it manifests no convincing seasonal incidence, and it does not occur in epidemic form. On the other hand, we know that acute facial palsy occurs as an occasional complication of certain virus infections, as well as quite often in the course of the Guillain–Barré syndrome. We remain uncertain whether the pathology in these cases is similar to that of Bell's palsy, and in the case of the Guillain–Barré syndrome the initial lesion is in fact likely to be both demyelinative and more proximal, in keeping with the general pathology of the condition.

It is truly remarkable that we really have no idea regarding the cause of this simple and ubiquitous condition, and are uncertain whether it is a result of ischaemia produced by exposure to cold, due to virus infection, or a variant of the allergic neuritis that can be produced by antigen-antibody reactions both in man and in the experimental animal. Bell's palsy takes its place among such mysterious conditions as neuralgic amyotrophy, retrobulbar neuritis, and paralysis of the long as well as the short nerve of the distinguished anatomist who gave it his name (Bell, 1829).

## Treatment

Only 10% of all patients with Bell's palsy are ultimately dissatisfied with the outcome. Recovery may not be electrophysiologically or even clinically complete, but it is functionally and cosmetically adequate. The value of radical treatment can be assessed only by its effect on this critical 10% of patients.

With regard to general management, the elderly patient may need to be reassured that the condition is not a stroke. Neither massage nor galvanism has any proved value in the treatment of this condition, and merely fill the physiotherapy department with people who should be at work. There is no earthly reason why cervical sympathetic blockade should be of value in Bell's palsy and no evidence that it is. Few of my patients have found the wearing of a wire splint or a piece of strapping on the face worth the trouble involved, but some with persistent paralysis have been helped by plastic surgery.

I have never referred a patient for surgical decompression, and in view of recent therapeutic developments I am unlikely to do so in the future. What is really important, where the paralysis is severe enough to prevent reflex blinking, is to protect the eye. If complete disappearance of reflex blinking leaves a good deal of cornea permanently exposed a patch should be worn over the eye and bland drops used several times daily. The reflex usually recovers fairly quickly, but if not the help of an ophthalmologist should be sought in management.

The validation of treatment with A.C.T.H. represents the most important therapeutic advance in the history of Bell's palsy. Dr. Taverner's careful observations (Taverner *et al.*, 1966) confirm my own less organized experience that really early treatment is highly successful. Recent controlled observations in Manchester have confirmed a similar though rather less dramatic efficacy of the same form of treatment in retrobulbar neuritis (Rawson *et al.*, 1966). With regard to Bell's palsy, a form of treatment that produces satisfactory results in 76 out of 77 cases leaves little room for improvement. The earlier treatment begins the better, and even on present evidence both efficiency and economy might best be served by side-stepping special investigations and regarding the condition as one to be treated at once by the general practitioner with a short course of A.C.T.H. Direct evidence of the effectiveness of prednisone

in the treatment of this condition is lacking, but it is possible that in really adequate dosage it would prove as effective as the less convenient pituitary hormone: a careful trial of early treatment with the oral preparation would be a very suitable project for the Research Committee of the College of General Practitioners.

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# Observations on Interpretation of Blood Alcohol Levels Derived from Analysis of Urine

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For many years it has been customary to derive blood alcohol concentrations from the analysis of urine, and despite the fact that the accuracy of such derivations has often been questioned British courts have been content to accept them as evidence in prosecutions for drunken driving, presumably on the grounds that such evidence was merely confirmatory and not vital. However, with the impending change in the law which will make it an offence to drive with more than a stated amount of alcohol in the blood, the reliability of the method of determining blood alcohol concentrations assumes much greater significance, particularly as the use of urine analysis has not been excluded (Ministry of Transport, 1965).

In the interpretation of blood levels derived from urine analysis two problems arise. The first concerns the accuracy of the analytical method used to measure the alcohol concentration in urine, and the second is related to the choice of the conversion factor for the derivation of blood level. In the case of urine analysis the accuracy of the Nickolls modification of the Cavett technique is well established, but the method is tedious, time-consuming, and critically dependent on the technical skill of the operator. Gas chromatography is free from these disadvantages (Chandela and Janák, 1960), and it is claimed that when used in combination with an internal standard and an integrator the alcohol content of a minute sample of either blood or urine can be measured accurately within five minutes (Curry, Walker, and Simpson, 1966). One purpose of the investigation to be described was to determine the accuracy of the gas chromatographic method.

In the case of the conversion factor no unanimity of opinion exists about the correct choice, and it was the second purpose of the investigation to examine the validity of the conversion factor of 1.33 recommended in the Report of a Special Committee of the British Medical Association (1965).

## Material and Methods

During the Christmas period of 1966 and the succeeding weeks one of us (D. V. F.) in his capacity as police surgeon obtained two specimens of urine at known time intervals and one of venous blood and sometimes one of capillary blood from 35 motorists suspected of driving under the influence of drink. The times at which the samples were obtained were noted and the samples themselves were divided into three equal parts for distribution to the suspect, the police laboratory, and this department. Anonymity was preserved by the designation of a reference number. Initially the samples were analysed both by the Nickolls (1960) modification of the Cavett technique and by the gas chromatographic technique described in detail by Curry *et al.* (1966) but modified to use a Poropak Q porous polymer bead column instead of a polyethylene glycol 400 column. The Poropak column offers the advantage of a much lower column bleed and elutes the water peak first after 30 seconds followed by ethanol at 1.5 minutes and n-propanol at 3.5 minutes. With polyethylene glycol, unless the column is carefully conditioned with water before use, a tailing water peak is produced which prolongs the analysis. Latterly the samples were analysed by the gas chromatographic method alone.

For chromatographic analysis the blood or urine sample was diluted with ten times its volume of an aqueous solution containing 24 mg. of propranol per 100 ml. to act as an internal standard. One microlitre (μl.) of this diluted sample was injected into the Poropak Q chromatographic column maintained at 170° C. The resultant peaks were detected by a flame ionization detector and the output signals fed into a digital integrator. Whenever possible the analyses were carried out in triplicate.

The accuracies of both the Nickolls and the gas chromatographic methods were tested by analysing samples of pure water and normal blood to which known quantities of alcohol had been added to achieve a range between 52 and 242 mg./100 ml. The purity of the absolute alcohol used in the preparation of standards was certified by the Laboratory of the Government Chemist using a refractive index method, as were the dilute solutions of standards. The solutions themselves were prepared by weight by means of an accurate analytical balance to achieve an alcohol content in distilled water which was between 30 and 32 mg./ml. In the preparation of the blood samples the alcohol solution was added by weight to a flask containing a known weight of blood equivalent to not less than 50 ml. The blood itself was magnetically stirred during and after the transfer for five minutes.

When the values for urine and the corresponding venous blood had been obtained it became obvious that additional information on urine: blood ratios would be an advantage. The

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