The following cases were studied on Wards 22 and 25 of the Edinburgh Royal Infirmary during the Phase III attachment to the Department of Clinical Medicine between May 7th and June 6th 1984. The necessary permission to present these cases was obtained from the physicians concerned.
NEURALGIC AMYOTROPHY
Case Report 

NEUROLOGICAL AMYOTROPHY

Name: Mr. L.G.
Age: 22
Occupation: Unemployed Electronics Technician
Interviewed by myself 7th May 1984.
Referred to Dr. R. Cull (Consultant Neurologist) by a psychiatrist at the psychotherapy O.P.D. of the Royal Edinburgh Hospital. Here it was observed that the patient was "unable or unwilling to move his (R) arm." The diagnosis of hysterical conversion syndrome was offered and the referral was made to exclude an organic cause.

PRESENTING COMPLAINT

1) Pain in (R) shoulder.
2) Reduced mobility and weakness of (R) arm.
3) Area of numbness over (R) deltoid region.
4) Change in shape of (R) shoulder

3 months

HISTORY OF PRESENTING COMPLAINT

Pain and weakness of (R) shoulder on and off for last 6½ years.
About 4 or 5 episodes during that time.
Most recent episode began 3 months ago. Consisted of-
1) Pain of sudden onset, dull aching, quite severe (keeping patient awake at night) site in - region of (R) deltoid muscle - back of neck on (R) side - lower down in (R) arm.
Dull pain became quite sharp on certain movements of shoulder.
Not relieved by analgesics from C.P.

Severe pain lasted 1 week, giving way to less severe pain now confined to the shoulder region and still present on admission.

3) Accompanying weakness, more gradual in development, prevented patient from raising arm above shoulder height.

No difficulty in gripping, either now or in previous episodes.

4) Area of numbness noted on lateral aspect of C8 arm, likened to dental anaesthetic. Not an accompaniment of previous episodes.

5) Change of shape of shoulder produced by loss of muscle bulk over previous 3 months.

Past episodes were described as consisting of pain and weakness in C8 shoulder lasting a few days. In particular,

1 year ago, during weight training session,

noted (a) C8 scapula “sticking out” at back
(b) C8 latissimus dorsi less bulky than the left
(c) Reduced mobility of C8 shoulder, concomitantly.

°Double vision at any time
°Migraines
°Occasional mild headaches, relieved by aspirin.
°Incontinence
°History of trauma, particularly to neck, prior to onset of symptoms.

**Family History**

Mother died, Ca. Stomach, when patient was age 12.

Father (age 60), 3 sisters (32, 26, 25) and 1 brother (30)

- all alive and well.

No family history of neurological disease
No diseases which run in family.

PAST MEDICAL HISTORY

(1) "Nervous breakdown" December 1981. Took two overdoses within a two week period in December (1) Few sleeping tablets (1) 70 Aspirin
A cry for help which brought him to the attention of the psychiatric services, and he has been attending psychotherapy a.o.d. at The Royal Ed. since early 1982. Psychiatric opinion was that he had "a depressive problem with roots in personality problems." The patient claims that his own emotional difficulties were due to an abnormal grief reaction during adolescence relating to his mother's death when he was 12. He feels he has now overcome these problems with the help he has received.

(2) Greenwich # 2 radius during car accident shortly before admission

No other injuries.

(3) "Diabetes mellitus" "epilepsy"

SOCIAL HISTORY

Lives with his father in Edinbrough. Sacked from "Ferrari's" 1 year ago following many absences associated with emotional problems outlined above. Two year engagement to girlfriend was terminated shortly afterwards. Started course at Leith Nautical College then finished soon after. Now unemployed. Previously very active with rugby, skiing and running but not since nervous breakdown.

DRUGS None

Smoking None

Alcohol Socially, or weekends only

Allergies None Known
Systematic Enquiry

Revealed nothing of note. No recent infections.

On Examination

A pleasant, co-operative apparently happy young man of small stature with C forearm in plaster.

CVS Pulse 60/min regular  B.P. 120/65 mm Hg

Chest Clear  N.A.D.

Abdomen No abnormalities detected (N.A.D.)

Neuromuscular System

Marked muscle wasting in C shoulder muscles.

especially  Supraspinatus, Infraspinatus

Deltoid, Latissimus dorsi.

C shoulder  N.A.D

"Faciulism  tremor  tenderness on palpation.

POWER

5/5 all groups in lower limbs

Upper limbs  C shoulder 5/5 all movements

C shoulder abduction 3/5  2/5 on full abduction

Abduction 4/5: action produced mainly by pectoral muscles.

C elbow extension 4/5

Flexion 4/5

C wrist and finger movements 5/5.

TONE  N.A.D

COORDINATION  Poor in C arm due to weakness.

REFLEXES

Preserved. Symmetrical and brisk.
Sensation

- Soft touch + pinprick sensation - absent
- Soft touch + pinprick sensation - diminished
  i.e. Loss of sensation in C8 axillary circumflex nerve
  distribution

Vibration sense → intact all groups
Proprioceptive sense

Cranial nerves - NAD

Impression
22 year old man presenting with 6½ year history of episodic pain, weakness, numbness and muscle wasting of C8 shoulder.

Diagnosis
History and examination most suggestive of NEURALGIC AMYOTROPHY. To exclude: Demyelination, Diabetes, spinal root involvement (disc prolapse), syphilis.

Investigations

1. Radiography - Oblique films of cervical spine to exclude cervical disc prolapse.
   Result - Minimal early osteophytic formation on the C side, at C4/5.
   The intra-vertebral foramina on the C side are normal.

2. Fasting Blood Glucose
   Glucose Tolerance Test → to exclude diabetic neuropathy, and
   Diabetic amyotrophy.
   Result - Diabetes not present.

3. Cerebrospinal fluid
   Cell count - One per cubic cm. (lymphocytes) Normal < 5 per cu.mm.
   Pressure - 16 cm H2O Normal 7-20 cmH2O
Glucose 3.1 mmol/l  Normal 2.8-4.2 mmol/l
Total protein 226 mg/dl  Normal 14-45 mg/dl
Y globulin 1.9 mg/dl  Normal 2.5 mg/dl

Microscopy - No organisms seen
Culture - No growth
Syphilis serology (VDRL and TRHA) - Negative.

6. No abnormalities found, particularly no evidence of multiple sclerosis (CSF protein normal or raised, with high Y-globulin)
No evidence of syphilis.

6. HAEMATOLOGY
6.1 ESR - 8. In particular a raised ESR might have suggested the possibility of polyarteritis nodosa which occasionally presents with polymyositis. Excluded here.

6.2 Anti-nuclear factor - ve. In systemic lupus erythematosus CNS changes are seen in half the patients, and may include depression and polymyositis. SLE excluded here.

6. NERVE CONDUCTION STUDIES and ELECTROMYOGRAPHY showed acute denervation of affected muscle, and changes consistent with Neuralgic amyotrophy.

6. Somatosensory evoked potentials measured from median and ulnar never did not positively establish diagnosis of neuralgic amyotrophy but helped exclude a demyelinating disorder.
Neuralgic Amyotrophy (otherwise known as Shoulder Girdle Neuromuscular Radiculitis, Brachial Plexus Neuropathy) is a localised neuritis of one or more nerves innervating the shoulder girdle and is a condition which often gives rise to diagnostic difficulties. The condition has been well documented before World War II, and was observed during the war, especially in the Near East (Spillane 1943; Low and Turner 1948). The syndrome was observed occasionally to follow after an immunisation or a non-specific infection, and often occurred in patients who were in hospital with an unrelated condition.

Pain is usually the initial symptom, usually acute in onset and intense for several days, gradually subsiding over several weeks. The limb becomes weak and as the pain settles, wasting is evident and may become severe. The muscles most often affected are

1. serratus anterior
2. spinati
3. deltoid
4. trapezius

LnG. presented with the characteristic pattern of pain and with wasting of spinati and deltoid. His history of "winging of the scapula" in the past is highly suggestive of previous involvement of the long thoracic nerve which supplies serratus anterior, and the muscles most commonly affected. Paralysis of serratus anterior leads to no deformity at rest, since it normally fixes the scapula to the chest wall. But when the patient exerts forward pressure with the arm against resistance the lower border of the scapula becomes winged, especially in the lower two thirds.

Involvement of the deltoid may be accompanied by sensory loss of the distribution of the axillary nerve (circumflex), as was evidenced in this case. This is the nerve most commonly affected.

More recent evidence has shown that other muscles, notably biceps and forearm muscles are less commonly involved. LnG. showed clinical evidence of involvement of biceps, and also triceps, not normally noted in this condition.

Nerve conduction studies and electromyography usually demonstrate slowing of nerve conduction and evidence of denervation. Brachial plexus involvement is discovered to be bilateral in 25% of cases, and very rarely there is mild lymphocytic pleocytosis or a rise in the C.S.F. protein (Weiskopf and Mattson 1969; Tsai et al 1972). In the case of LnG., C.S.F. protein was within the normal range and the white cell count was not abnormal.

The aetiology of the condition is as yet unknown and the pathology can seldom be studied, but an identical syndrome can follow seven to ten days after the injection of a foreign serum. More
Recent experience has demonstrated that in more than one third of cases there is no antecedent history of illness, infection or trauma. In some families a recurrent tendency to suffer from neuromyotonia may be transmitted by dominant inheritance (Taylor 1960, Geiger et al 1974, Bradley et al 1975). There was no family history in the case studied, neither did the episodes appear to be related to preceding infection or trauma.

Although the initial pain is very severe and paralysis and wasting are often marked, recovery of muscle power is usually excellent, beginning within one or two months, but complete return of function may take up to three years or more (Tsai et al 1972) only occasionally is paralysis permanent, and is more likely in older patients, especially those with paralysis of deltoid or serratus anterior.

Firm diagnosis of this condition is important as it prevents unnecessary invasive investigations. Treatment is symptomatic, powerful analgesics being required at the onset of an acute episode. Steroids are ineffective in shortening the duration of an attack, although there is controversy as to whether administering steroids at the onset would abort paralysis. Regular passive movements (avoiding overstretching) should be encouraged with active exercise advocated as soon as possible, at first assisted by a physiotherapist if necessary. There is possibly some value in regular electrical stimulation of paralysed muscles. Sclaram suggests that square wave stimulation of paralysed muscles at a frequency of 40 stimuli/minute is of some value.

One interesting aspect of this case is that the initial diagnosis offered was of hysterical conversion syndrome. The central feature of this syndrome is a loss or alteration in physical functioning apparently due to a physical cause, but which is in fact a manifestation of some underlying psychological conflict or need. Almost any physical symptom can be produced, but most commonly the symptoms are suggestive of neurological disease. In particular, paralysis and anaesthesia, both features of this case, are often manifested. The diagnosis cannot be made simply because no evidence of organic cause for physical symptoms can be found. Studies by Slater (1965) and Medsley (1976) both showed that when cases diagnosed as hysterical conversion syndrome were followed up high rates of overt organic disease were later discovered. The case of L.G. demonstrates this principle quite nicely. Although the patient had a previous psychiatric history, including deliberate self poisoning, and although the psychiatric opinion concerning his personality was that he was “an old adolescent who has not yet fully or satisfactorily made the transition into adulthood”, relatively simple medical investigations soon made it clear that the presenting symptoms were organic in nature and not psychogenic.
After eight days in hospital L.C. was told that he had an inflammation of the nerves supplying his shoulder muscles and that function would probably return over the next few months, though a 100% guarantee of this could not be given. As function was beginning to return he was encouraged to use the arm as normally as possible. His G.P. was informed of the diagnosis, so that adequate analgesic relief could be supplied for any further episodes. The referring psychiatrist was also informed of the organic nature of the patient's symptoms.
REFERENCES


Seddon H.T. Surgical disorders of peripheral nerves. Edinburgh and London.
HYPERTHYROIDISM
HYPERTHYROIDISM

Name: Mrs. J. B.
Age: 34
Occupation: Housewife; works part-time in a children's nursery
Interviewed by myself 20/5/84
Elective admission 20/5/84 to Ward 25.

Referred to Dr. W.J. Irvine (Consultant Physician) by GP, who on account of her diffuse goitre, marked tremor and other symptoms described Mrs. J. B. as having obvious thyrotoxicosis.

PRESENTING COMPLAINTS

1. Swollen neck
2. Uncontrollable shaking hands
3. Sweating and heat intolerance
4. Agitation
5. Weight loss

HISTORY OF PRESENTING COMPLAINT

About 1 year ago - noticed that she "could not slow down."

Hyperactivity — tiredness, but was unable to sit still.

Noticed increased irritability with husband and children.

Seven months ago - first noticed fast tremor of hands which became progressively worse over ensuing months.

Lost 1/4 stone in weight over next four months despite eating normally.

Periods - duration became only 1½ days instead of previous 4.

Three months ago - noticed that she was unable to wear
a tight collar. At this stage her sister (a nurse) noticed the
grip and J.B. went to see her C.S. (Feb 1984)
Symptoms in Feb. now included
- marked heat intolerance
- marked sweating, esp. face and palms
- palpitations; fast pounding heart, accompanied by loud
pounding sound in her ears.
- marked irritability
- extreme tiredness.

Was seen by Dr. Irvine in March and started on propanolol
(40mg t.d.s) having ensured there was no history of asthma.
The dose was increased on two further visits to the O.S.B.
finally becoming 80 mg t.d.s. Symptoms were still present though
less marked.

PAST MEDICAL HISTORY
Sterilization 1978 Royal Infirmary of Edinburgh.

Eczema since 1972.

*Diabetes *IHD *Hypertension *asthma *anaemia *blood transfusions

tum 2 t o

DRUGS

Propanolol 80mg t.d.s.

Potassium iodide 60mg t.d.s.

Smoking 20/day for 13 years

Alcohol very occasionally

Allergies none known

FAMILY HISTORY

\[
\begin{align*}
\text{F} & \quad \text{M} & \quad \text{S} & \quad \text{B} \\
\end{align*}
\]

alive and well

Sister has history of

"thyroid trouble" ? simple goitre.
No family history of Diabetes / Ischaemic heart disease
No diseases which run in family.

**SOCIAL HISTORY**

Lives in house in Edinburgh with husband and two children.
Husband self-employed.
No problems at home.

**SYSTEMATIC ENQUIRY**

Nothing else of note elicited

Periods became regular every 28 days and lasting 4 days since beginning treatment with propanolol.

Frequency of bowel motions not increased.

**On Examination**

Agitated appearance, constantly fidgeting with hands and moving feet. Euphoric - smiled and laughed nervously throughout interview. Hands - warm and moist

**Neck** - Diffuse, moderately enlarged, symmetrical goitre.

Smooth edge, uniformly soft consistency.

Freely moves upwards on swallowing

- retrosternal extension
- thrill

Soft bruit heard over ® lobe.

- associated lymph nodes.  — lymphadenopathy elsewhere

**CVS**

Pulse 72 / min regular in time and force. No atrial fibrillation.

BP 100/50 mm Hg

- murmur, particularly none associated with aortic valve.

- carotid bruits.

**Chest** Clear. NAD.

**Abdomen**

- Appendix
- Striae

Soft. NAD.
Nervous System.

Tremor - fine, exaggerated physiological tremor. Both hands.
Sheet of paper laid over outstretched hands - obvious continuous fine movements.
Slight lid retraction - patient claims that her eyes have always been open wider than most peoples from being a child.

No "lid lag" elicited.

No other abnormalities detected.

Tone | Cranial Nerves | Normal
Power | Normal Sensation
Co-ordination | Reflexes - symmetrical and brisk.

Locomotor System

NAD - legs normal - no pretibial myxoedema.

Impression - 34 year old previously fit married woman presents with one year history of thyrotoxicosis. On admission, undergoing medical pre-operative preparation for subtotal thyroidectomy.

Investigations

Full blood count
ESR
Urine and electrolytes
Liver function tests
Serum calcium

Radiography: Chest X-ray + Thoracic vixx - Normal

All within normal ranges
ECG: Normal. Sinus rhythm 75/min regular.
Indirect laryngoscopy:
E.N.T. specialist opinion sought.
"Larynx and pharynx both healthy.
Normally mobile. No pooling of saliva.
(+) vocal cord slightly thickened"

Thyroid Antibodies (17/4/84)
Thyroid cytoplasm   Gastric parietal cells 
Thyroid microsome Anti-nuclear factor
Thyroglobulin Smooth muscle
mitochondria

Æ Auto-immune thyroiditis excluded.

Thyroid Scan (14/3/84)
Oral tracer dose of $^{131}$I given. Thyroid gland uptake measured four hours later, by "counter" over neck.
Result - uptake was raised at 52% (normal range 10-40%)
Isotope scan confirmed that J.B. had a diffuse enlargement of the thyroid with uptake throughout the gland.

Serum Thyroxine = 236 nmol/l (Normal range under 65 - 70-150 nmol/l)
Serum TSH = < 0.8 mU/l (Reference range up to 5.7 mU/l)
Æ T4 elevated in presence of reduced TSH. Typical biochemical picture of thyrotoxicosis.

Serum TSH levels in response to TRH stimulation - not tested as diagnosis of thyrotoxicosis not equivocal.
The investigations and drug preparation were sufficient for the operation of sub-total thyroidectomy. (See following commentary.)

The operation was carried out on 22/5/84 the patient having been transferred to Ward B. Following the operation the patient developed a wound haematoma, without any respiratory symptoms, and was resuscitated under general anaesthesia for evacuation of the haematoma. The following day the wound was fine, and the patient was making a good recovery. Her only complaint was of discomfort on swallowing, and already she could feel the benefits of the operation, feeling much less anxious within herself. She appeared calmer and more relaxed and said that she felt "a different woman", "back to her old self."

Careful follow-up of Mrs J.B. will now be required to monitor for recurrence of symptoms, and indeed for the symptoms of hypothyroidism, one of the relatively common risks of the operation. Assessment of the adequacy of her control will be made primarily on clinical grounds e.g. should the patient begin to complain of sensitivity to the cold, constipation, weight gain, tiredness and forgetfulness, hypothyroidism would be strongly suspected. Assessment of the function of the patient's thyroid can also be assisted by measuring the total T3 and T4 in a sample of blood. In the majority of cases of hyperthyroidism this commonly used in vitro test of thyroid function will give unequivocal confirmation of the diagnosis. Further confirmation in hypothyroidism could be gained from the finding of a markedly elevated TSH. Abnormal TRH test are useful in diagnosing borderline states of hyperthyroidism. i.e. a lack of response of serum T.S.H. to intravenously administered T.R.H.
Since parathyroid insufficiency is also a significant complication of
of the operation, the calcium levels in the blood taken for T3 and
T4 analysis could also be usefully measured. The normal range of total
calcium is 2.12 – 2.62 mmol/l, but this may vary with high or
low albumin levels, in which the ionized calcium level, which is the
important fraction, remains within the normal ranges. Hypoparathyroidism,
as a transient phenomenon, is common after partial thyroidectionomy, but
may occasionally be more prolonged. In adults the resultant tetany
may present with symptoms of tingling in the hands, feet, and
mouth, and less often with painful carpopedal spasm. Stridor and fits
may also occur but are rare.

Assays of Parathyroid hormone itself are now available and would
assist in diagnosing parathyroid insufficiency in this instance.

Where tetany follows partial thyroidectionomy, remaining parathyroid
tissue usually hypertrophies to compensate. Until this occurs intravenous
calcium gluconate may be required to control the tetany. If all
of the parathyroid tissue has been removed, long term replacement
therapy with calcium (usually 1.25 mg daily, i.e. one tablet) can be
used to maintain a normal calcium level.
Apart from displaying the classic signs of thyrotoxicosis, the case of Mrs. J. B. is interesting in that it allows consideration of the various ways of tackling toxic diffuse goitre (primary toxic goitre). The options open are:

(a) 
anti-thyroid drugs
(b) 
surgery
(c) 
radio-active iodine

(3) Carbimazole or methimazole, initially in conjunction with β-adrenergic blocking agents such as propranolol, may be the therapy of choice or may be used to prepare the patient for surgery, where antithyroid drugs are used alone, their use should be continued for at least one year and only in patients who do not have a large goitre. Such selectivity will produce lasting remission in just under 50% of cases. (Potassium perchlorate should be avoided where possible because of its association with blood dyscrasias such as red cell aplasia, which is usually lethal.) The younger the patient the better the chance of complete remission with antithyroid drugs (Leave “surgery” 1969, 1xv, 649), but if this fails operation should be advised. McChintock (“surgery” 1969, 1xv, 700) suggested that the main value of anti-thyroid drugs is in making surgery a safer procedure.

(6) Surgery provides a cure by reducing the mass of overactive tissue. It is the treatment of choice when the patient is considered too young for radioactive iodine therapy (i.e. during reproductive years) or where antithyroid drugs have been tried and failed, or have produced hypersensitivity reactions. Thus, where there is a large goitre in a thyrotoxic patient below the age of forty, surgery should be considered in preference to a prolonged treatment with antithyroid drugs, which when withdrawn may allow the condition to return. Particularly in women, a thin thyroidectomy scar may be more cosmetically acceptable than a large goitre. Advantages of surgery are that the cure is rapid and the cure rate is high if surgery has been adequate. The disadvantages are that a recurrence of thyrotoxicosis occurs in about 5% of patients, and every operation carries a morbidity and a mortality. However, with a suitable surgeon and adequate preparation mortality is negligible. The only significant complication due to operation is parathyroid insufficiency.

(6) Radioactive iodine is the treatment of choice for patients over the age of forty, for women who have been sterilised or for those whose life expectancy is reduced for whatever reason. The risk of developing thyroid cancer or leukaemia following “I therapy has been shown not to be increased, and there are no complications other than hypothyroidism in the years following therapy. Because of
this complication however, regular review is essential.

These then are the indications for the various forms of therapy. In practice treatment for a patient is often arbitrary, and the patient may express a particular wish once the various possibilities have been explained.

In the case of Mrs. J.B., radioactive iodine was not offered, since the clinician considered her unsuitable because of her youth. She is thirty-four. However, she was sterilized six years ago and, theoretically at least, would therefore, been a suitable candidate for radio-iodine therapy. Perhaps this is an indication that there is still uncertainty about the long-term carcinogenic effects of radio-iodine therapy in the minds of clinicians, or simply that the risk of hypothyroidism following this form of therapy was greater than the risks associated with medical and surgical forms of treatment.

When offered the choice of medical or surgical treatment the patient expressed a strong preference for surgery, because of the undesirability, for her, of taking tablets on a long-term basis. Indications for surgery in her case were that she had a large goitre and was below the age of forty.

**Pre-operative preparation**

The case of Mrs. J.B. is exemplary in demonstrating the principles of pre-operative preparation.

Adequate medical preparation is vital before surgery on any toxic thyroid. Riddell (British Journal of Surgery, 1970) suggested that this should always include laryngoscopy to assess the state of the vocal cords, because of the risk of recurrent laryngeal nerve paralysis.

Whilst many sources advocate the use of carbimazole (30-100 mg) may 8-12 weeks before operation, Riddell states that anti-thyroid drugs increase congestion and add to the risk of haemorrhage during surgery. (B J S, 1970). The policy he advocated was to make the patient euthyroid with propranolol (80 mg t.d.s) for 10 days before operation. Propranolol acts in two ways: (i) by blocking the peripheral adrennergic effects seen in hyperthyroidism, and (ii) by inhibiting the peripheral de-iodination of thyroxine to tri-iodothyronine, the active form of hormone.

It does not reduce the output of hormone or the circulating pool of thyroxine and since it only acts for six hours, a return to the hyperthyroid state will quickly follow any step in treatment. Thus, an omission of a single dose before operation can have drastic consequences, causing severe hyperthyroidism, or even
Thyrotoxic crisis. This creates the need for good communication between the medical and surgical doctors handling any case of thyrotoxicosis requiring surgery.

Propamol should be continued for seven days after surgery. Before surgery the adequacy of β-blockade should be demonstrated by exercise tolerance (monitoring onset of pulse rate). Some patients are very resistant to β-blockade by propranolol and in these, the high doses required before surgery should be reduced immediately afterwards when there will be a drop in serum levels of T4 and T3.

Pre-operative investigations should always include
(a) Indirect laryngoscopy - a symptomatic cord palsy exists in 3% of cases with no previous thyroid surgery.
(b) Thyroid antibodies - to exclude presence of autoimmune thyroiditis
(c) Serum calcium estimation
(d) Thyroid scan to provide information about toxic nodules, if present.

Complications of Subtotal Thyroidectomy

1. HAEMORRHAGE - Despite preparation with a drug regime intended to decrease the risk of haemorrhage Mrs. J.B. developed a wound haematoma several hours after her operation, and this is a fairly common post-operative complication. A tension haematoma, developing deep to the cervical fascia, can produce respiratory obstruction by producing laryngeal oedema, and when this happens it may be necessary to open the wound on the ward to relieve tension before taking the patient to theatre. In Mrs. J.B.'s case there were no respiratory symptoms and the second trip to the theatre was uncomplicated.

2. PARATHYROID INSUFFICIENCY - Usually present dramatically 2-5 days after operation, or a patient with marked hypocalcaemia may be asymptomatic.

3. THYROID INSUFFICIENCY - requires long-term follow up. It is often very mild in presentation and should always be looked for with a high index of suspicion.

4. THYROTOXIC CRISIS (STORM) This is an acute exacerbation of hyperthyroidism characterized by tachycardia, high fever, restlessness and delirium. This is now extremely rare because of the adoption of good medical pre-operative preparation, as described in the above account.
Case 2  HYPERTHYROIDISM


REEVE T.S. et al (April '69) Thyroidectomy in the management of thyrotoxicosis in the adolescent

Surgery 65, 694-9

McCINTIOCH J.C. An appraisal of the current methods of treatment of toxic goitre.

Surgery 65 : 700-7 (April 1969)
INTRACTABLE ANGINA
Case Report

INTRACTABLE ANGINA

Name: Mr. J. M.
Age: 70

Occupation: Retired 5 years ago. Previously plumber and joiner.

Interviewed by myself 15th May 1984
Admitted 16th May 1984 Ward 22 waiting night at 11 pm, from Casualty Department. Waiting Physician, Professor Oliver.

PRESENTING COMPLAINT

Chest pain - began 7 hours ago.

HISTORY OF PRESENTING COMPLAINT

Pain came on at rest at 4 pm in afternoon.
Located (with one finger) on left side in 3rd intercostal space.
Radiating down left arm to fingertips and up into neck.
Also radiating into left side of neck.

Constant, severe, feels "like cramp."
10 GTNs produced only slight relief as did morphine given by CR at 7 pm.

Swearking and nausea associated with pain.

- vomiting
- dyspnoea
- palpitation
- ankle oedema.

J.M. claims never to have been entirely free from chest pain since third heart attack in 1983.
1968. Began to suffer from angina. Great pain on exertion, gradually became more frequent and more severe.


1974. Coronary artery bypass graft (3 vessel disease) plus left ventricle aneurysmectomy.

For next nine years totally asymptomatic. Continued to smoke 15 cigarettes/day.

1982. Inferior M.I.

Followed by recurrence of angina, same as previously, and mild left ventricular failure, managed with diuretics.

1983 (September) Inferior M.I.

1984 (April) Radionuclide ventriculogram. Ejection fraction = 22%

Chronic bronchitis - of many years standing. ODM & BP

DRUGS

G.T.N. p.r.n. + Nimoderm plaster one/day.

Ismo 20 t.d.s.

Fusenide one per day

Slow K b.d.

Ampicillin - for chest infection, recent onset (previous year)

Smoking 10-15/day for all his life until November '83 following third heart attack. Restarted smoking cigars occasionally since March '84.

Drinking Moderate

Allergies Asprin, produces waterbrash.

SOCIAL HISTORY

Lives in flat with wife. No stairs to climb.

Manages to do a little gardening.
FAMILY HISTORY

Ischaemic Heart Disease

Angina
Angina
Several M.I.s
Heart valve disease
- had Rheumatic fever as a child

5 siblings.
No others with heart disease

All alive and well.

ie. strong family history of ischaemic heart disease.

SYSTEMATIC ENQUIRY.

Cvs. See presenting complaint.

Rs.
Cough
Spitum - green/yellow
on and off for many years.
Acute exacerbation in March, still
persisting. Antibiotics prescribed.
Coughs up occasional blood stained sputum.
Wheezes on exertion.

GIT. NAD.

GU. NAD. (frequency produced by diuretics only)

CNS. Weakness in arms and legs last year. (September 1983)
Associated with talking of ADRAT. Symptoms relieved when
drug withdrawn.

Hearing declined over last 5 years.

Nil else.

Cvs. See presenting complaint.

Also: since September '83 chest pain has been constantly
present, becoming worse on exertion. More severe exertional pain relieved by GTN but more constant pain never relieved. Sometimes pain becomes more severe at rest and may wake patient from sleep. Again this more severe pain is always relieved by GTN.

2) Occasional Paroxysmal Nocturnal Dyspnoea - occurs about once/month.

3) 3 year history of intermittent claudication.

Pain in both calf on walking 200 yards. Relieved by rest.

On Examination

Tall, rather obese man, obviously in discomfort.

Finger clubbing (first noted in 1973; pneumoconiosis associated with work in mines)

C/S

Pulse 92/min regular bide and force

Br 150/90 mmHg. Apex beat 5cm I.C.S. Mid clavicular line.

JVP not elevated, no ankle/sural oedema

Heart sounds I and II added sounds murmurs thills.

Peripheral pulses

@ femoral built

@ posterior tibial and @ dorsalis pedis absent

@ foot cooler than @

All pulses present on @ side.

RS Trachea central

Chest clear. No respirations.

Abdomen

Scars of Coronary Artery Bypass Grafting (1974)

Nil else.
Impression: 70 year old smoker and ex-miner with chronic obstructive airways disease and 16 year history of ischaemic heart disease presenting with severe chest pain radiating into arm and neck.

Diagnosis: ? Myocardial infarction

? Severe myocardial ischaemia

Confirmation of the diagnosis was made by obtaining serial E.C.G.s and sending off blood samples to clinical chemistry for serial enzyme studies.

E.C.G.s: ST elevation in leads V2, V3 and V4 = acute infarct, anteroseptal most likely.

Evidence of old myocardial infarct = Q wave in II, III and AVF

Enzymes

<table>
<thead>
<tr>
<th>9am</th>
<th>16/5/84</th>
<th>17/5/84</th>
<th>18/5/84</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspartate aminotransferase</td>
<td>127</td>
<td>193</td>
<td>172</td>
</tr>
<tr>
<td>Lactate dehydrogenase</td>
<td>462</td>
<td>1068</td>
<td>1256</td>
</tr>
</tbody>
</table>

Thus on the basis of the history, serial ECGs and serial enzyme changes the diagnosis of Mr J.M.'s fourth myocardial infarct was made.
Mr. J.M. presented with chest pain, one of the most common presenting symptoms on any male medical ward in the UK. As an obese 70-year-old male smoker with a strong family history of ischaemic heart disease behind him he had managed to collect several of the risk factors for atherosclerotic vessel disease. He is unusual in that he appears to have intractable angina. He is never free from chest pain, always located in the left side of his chest and worsened by exercise. I wish to consider what treatment, if any, might be offered to help this particular man.

In March of this year it was decided to test Mr. J.M.'s left ventricular function with a view to re-investigating his coronary arteries for possible surgery. He had already had coronary artery bypass grafts performed in 1994 which were successful and gave complete relief of symptoms for nine years. Re-operation is possible, and is indicated in patients with recurrence of their angina symptoms due to graft closure or progression of disease in unoperated arteries, providing the distal vasculature and the left ventricular function are still adequate for surgery. The mortality for this procedure is only slightly higher than that in the initial operation, and relief of angina occurs in about two-thirds of patients.

Investigation of ventricular performance is by radionuclide ventriculography (RNV). This involves intravenous injection of a radiotracer, usually Technetium 99m, which delineates the great vessels and chambers of the heart. Computed imaging provides information about right and left ventricular ejection fractions, ventricular volumes, and ejection and filling rates. In Mr. J.M. RNV showed a dilated left ventricle contributing poorly with septal akinesis. The resting ejection fraction was shown to be 23%, the normal for patients over 65 without arterial disease being just over 50%.

This very poor result excluded any further possibility of surgery since adequate left ventricular function is a necessary prerequisite.

Medical treatment of unstable angina, which is in effect what Mr. J.M. has, is essentially three-pronged. Nitrites, beta-blockers and calcium channel blockers can all be used. Mr. J.M. is already using GTN in the sublingual and dermal forms, yet they leave him with residual pain.

Beta blockers are contraindicated in the presence of chronic obstructive airways disease, which Mr. J.M. has had for many years, because of the added risk of bronchospasm to an already compromised respiratory system. However, since there are few other options open in this case it was decided that pulmonary function tests ought to be done to assess the extent of the respiratory disease.
The results were as follows:

<table>
<thead>
<tr>
<th>Ventilatory Capacity</th>
<th>Predicted</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV₁</td>
<td>3±0.5</td>
<td>1.2</td>
</tr>
<tr>
<td>Vital Capacity</td>
<td>3.9±0.65</td>
<td>2.7</td>
</tr>
<tr>
<td>FEV₁ / VC %</td>
<td>76±5</td>
<td>44</td>
</tr>
<tr>
<td>Residual Volume</td>
<td>2.2±0.4</td>
<td>4.44</td>
</tr>
</tbody>
</table>

These results clearly show a marked obstructive ventilatory defect with a raised residual volume. Gas transfer incidence was normal, and there was a clear response shown to salbutamol.

In addition to this well demonstrated respiratory disease Mr J.M. has had a 3 year history of intermittent claudication. Both blockers have a direct effect on peripheral blood vessels and may exacerbate intermittent claudication, and so again they are contraindicated in this case.

Adalat (Nifedipine), whilst probably indicated in this case has been tried in Mr J.M. and had some unusual effects, including weakness in his arms and legs which makes them unsuitable for him. The history is rather unusual and atypical for this particular drug, and in my opinion it would be worth trying the drug again to see what effects it had a second time. Alternatively there are other calcium channel blockers, such as Lidoftazine, which could be tried in this man.

These then are the main lines of treatment in this condition yet because of the extent of cardiac pathology and coexisting disease they are not suitable for this patient.

Several recent American journals suggest the use of aspirin in unstable angina since it has been postulated that platelet aggregation at the site of critical obstruction in the coronary vascular bed may intensify ischemia. These considerations led to a large randomised trial in America lasting 12 weeks in which men with unstable angina took aspirin, a potent antiplatelet. One aspirin daily resulted in a 50% decrease in mortality and non-fatal myocardial infarcts. (Lewis and others Circulation 66 part II : 17. 1982)

The evidence for the efficacy of aspirin is quite convincing. However, it was noted that Mr J.M. is "allergic" to aspirin, the only thing he is allergic to. It gives him waterbrash.

The outlook for this particular man with such extensive vessel disease is not good. It seems also that he will have to
endure his intractable angina for the rest of his life since there is nothing else left to offer him. When I last spoke to him he told me that he had been told nothing of what was likely to happen to him, or his pain. He had however come to his own conclusion that he was never likely to be free from chest pain again.
Case 3: Intractable Angina


Protective effect of 324 mg aspirin daily in men with unstable angina: Results of a VA Co-operative Study

Circulation 66 (Part II): 17 1982 (Moscow)
"INCIDENTAL" BRONCHOGENIC CARCINOMA
"INCIDENTAL" BRONCHOGENIC CARCINOMA

Name: Mr. F.K.
Age: 73
Occupation: Ex-roofer and laborer. Retired age 65.
Interviewed by myself 8th May 1984
Admitted 8th May 1984 Ward 22 waiting night at 8pm from Casualty Department. Waiting Physician: Dr. Horsley.

PRESENTING COMPLAINT

Blackouts x 2 Today (1) About 1:30pm in the street (2) 5:30pm in Casualty Department.

HISTORY OF PRESENTING COMPLAINT

Went to "Lochrie Arms" public house this morning (11:45 am) drank about four halves of whiskey.
Remembered leaving pub on his way home (2-30pm) then collapsed in the street.
Mr. F.K. has no memory of this. There was no warning.
Remembered finding himself in Casualty Department.
In Casualty, found to have fractured left surgical neck of humerus, and lacerated skin above left eye.
Given a collar and cuff for the fracture.
On standing up to go home (5:30pm) he was seen to go extremely pale and when instantly re-examined was found to be pulseless. Mr. F.K. sat down, became very breathless and began to lose consciousness.
Following two blows to the chest by the Casualty Officer his pulse returned.
An E.C.G. taken at this time showed a sinus bradycardia of 40/min.

Again the patient has no memory of this second episode. He was admitted to ward 22 to investigate further for a cause of his fall.

**DRUGS** None on admission

**Smoking** 15/day for many years

**Alcohol** Usually two pints per day

**Allergies** None Known

**PAST MEDICAL HISTORY**

1. **# 1** Neck of femur following a fall on the ice during winter 1982 - Princess Margaret Rose.

2. Bilateral ectropion repair 1982 Edinburgh Eye Pavilion

3. o Diabetes mellitus o T.B. o Epilepsy o Hypertension

   o Rheumatic Fever

**SOCIAL HISTORY**

Lives alone in a house in Lochbin Place, Edinburgh. Wife lives in Canonnmill. They are not divorced but have “gone separate ways.” Home Help comes three times a week.

**FAMILY HISTORY**

Accidental death

+35 o 100  

Alive and well - ages not known.

Five daughters - ages not known

No sons
Mr. F.K. has lost contact with both of his sisters and three of his five daughters.

Family History of Ischaemic Heart Disease or Diabetes.

**SYSTEMATIC ENQUIRY**

**CNS**
Denied chest pain, breathlessness, palpitations and ankle swelling.

Pain in both calves on walking ½ mile - Mr. F.K. did not know how long this had been present.

**RS**
Smokers cough

Copious clear sputum, especially in the morning many years.

Occasionally blood-stained sputum - once every few months.

Nil else.

**GI**

Appetite good

Weight loss - 1 stone over last 2 years.

Heartburn - once per night, relieved by antacids.

*Vomiting*  *Melena*  *Alteration in bowel habit.*

**CNS**

No headaches or double vision. No vertigo.

Nil else.

**GU**

Frequency x 5/day  Norturia x2/night

*Haematuria*  *Dysuria*

Nil else.

**LMS**

*O Surgical neck of humerus.*

No joint pain suggestive of severe arthritis.
On Examination

Pale, tided looking man in obvious pain from shoulder. Slightly confused, disoriented in time, but not in place or person. ? Early atherosclerotic cerebral involvement.

Poor historian - very vague. Alcohol detected on breath.
Slight finger clubbing. Lymphadenopathy detected.

- Gait
- Cyanosis
- Jaundice

CNS

Pulse rate 68/min, regular, tine and force.
BP 140/90 mm Hg

Apex beat 5 th I.C.S. Mid clavicular line. JVP - not elevated. Heart sounds I and II. No added sounds, No murmurs. Peripheral pulses all intact.

RS.

Trachea central.
Expansion R = L

↓ Breath sounds. ↑ Vocal fremitus at L base.

- Crackles
- Rhonchi.

Abdomen

Dull to percussion 3cm below costal margin. No masses, no tenderness, no guarding.

- LKKS palpable
- Bowel sounds present.

PR - not done, because of risk of inducing further arrythmia.

CNS - NAD.
Impression: 73 year old smoker presents with two episodes of loss of consciousness, the second seem to be associated with a sinus bradycardia.

Differential Diagnosis

(a) Stokes Adams attacks
(b) Asymptomatic myocardial infarct
(c) Postural hypotension

Investigations

(1) 24 hour ECG tape
(2) Serial ECGs (3 days)
   Serial enzyme assays - AST and LDH (3 days)
(3) Monitor lying and standing blood pressures

Also -

- Urine and electrolytes
- Full blood count with platelets
- E.S.R.
- Plasma glucose (fasting)
- Thyroxine
- Chest X-ray

Results

(1) 24 hour ECG tape. No abnormalities detected.
   Sinus rhythm throughout.
(2) Serial ECGs and enzymes. No evidence of infarction.
(3) BP monitoring. No evidence of postural hypotension.
Also plasma glucose < 5.5 mmol/l. No evidence of diabetes or hypoglycaemia.

Thyroxine was noted to be at lower end of normal
Haematology showed a very mild iron deficiency anaemia with a raised E.S.R. of 62.

Radiography - Mr F.K. had an X-ray film of his left shoulder taken, and on the day after his admission it was noted that there was a homogenous oval opacity about 3cm in diameter in the left upper zone of the lung field. Further PA and lateral views were taken and the opacity was confirmed to be in the left upper lobe with an appearance compatible with bronchogenic carcinoma. No hilar lymphadenopathy was seen.

His history of occasional haemoptysis and the findings of mild finger clubbing on examination, along with decreased breath sounds and increased vocal fremitus on the left side make the diagnosis of bronchogenic carcinoma more likely.

Samples of sputum were sent for cytology and a bronchoscopy was arranged. Cytology was normal. No lesions were visualised on bronchoscopy but under X-ray screening bronchial biopsies and brushings were taken from the site of the lesion in the upper lobe. No malignant cells were identified by the pathologists.

Mr F.K. remains on Ward 22.

See commentary for further discussion.
The case of Mr. F.K. is interesting for several different reasons.

The patient presented with a story of loss of consciousness. The causes of weakness, fainting, and disturbed consciousness are legion (there are over 30 listed on one page of "Harrison's Principles of Internal Medicine") but attention was focused on bradycardia as being a cause in this instance, since an E.C.G. in casualty following an acute episode showed a sinus bradycardia of forty beats per minute. Investigations for other common causes, including anaemia, secondary to an infection, and hypoglycaemia, were commenced. While the results of these tests were being awaited an X-ray film of Mr. F.K.'s shoulder was re-examined.

The fracture of the neck of the humerus which he had sustained when he blacked out on the fire occasion was self-evident. But on closer inspection of the adjacent lung field, an opacity in the upper lobe on the left side was noticed - the "incidental" bronchiogenic carcinoma of the title. It is "incidental" in the sense that Mr. F.K. would have been sent home without it ever having been noticed, had he not collapsed a second time as he was preparing to leave casualty, and therefore the collapse which secured his admission to ward 22. While this is not intended to be a criticism of overworked, underpaid casualty officers, it provides a good illustration of the need for thorough examination and scrutiny of available evidence, even that which may seem purely incidental to the main issue.

For Mr. F.K., the cause of his blackouts remains obscure. The normal 24 hour E.C.G. tape provided no evidence of intermittent Stokes-Adams attacks, or other bradycardiac rhythm. Therefore the bronchiogenic carcinoma is thought to be present. The possibility of a cerebral metastasis as a cause of his altered consciousness must be strongly suspected, and a C.T. scan may provide confirmatory evidence of this.

The investigation and possible treatment of the lung lesion was now the medical problem of prime importance in this case. Bronchoscopy and cytology however did not confirm the presence of any malignancy and reinvestigation may be required. Transbronchial or percutaneous lung biopsy, or mediastinoscopy can be used to confirm the diagnosis. Further cytological examination of sputum can be attempted, and percutaneous needle biopsy could be resorted to, to obtain a positive histological diagnosis in such a peripheral tumour.

The question then remains of what should be done with this man. Assuming a positive diagnosis is made and a cell type for the humerus is identified, surgical or radiotherapy treatment...
might be considered. Pneumonectomy or lobectomy offer the best prospect of survival but the only eligible cases for this treatment are those in whom the tumour is located at an early stage and in whom pulmonary function is adequate. Only about 20% of cases fall into this group, and Mr. F.K. is unlikely to be one of them. Although his pulmonary function has not been tested formally, he has been a smoker all his life and has a history suggestive of long-standing chronic bronchitis.

Radiotherapy can eradicate small tumours and in this case there appears to be no metastasis elsewhere in the lungs (NB. The possibility of cervical metastasis has already been mentioned). Radiotherapy is more commonly used, however, to relieve the distress of complications such as superior vena caval obstruction or chest wall invasion.

The use of cytotoxic drugs is still at an experimental stage. It is said to be able to increase the median survival of highly malignant cell types from 3 months to one year. The outlook in all forms of bronchogenic carcinoma is not good. The average period of survival after diagnosis is less than a year.

The form of treatment, if any, for Mr. F.K. will be decided once the diagnosis has been firmly established. It is most likely that radiotherapy will be attempted.

The question of where Mr. F.K. goes from hospital has also been raised. He lives alone in a house in Edinburgh, he and his wife having gone their separate ways. One of his five daughters still maintains contact with him but is unable to take him in herself, because of her own family commitments, but is concerned that he should not return to his previous situation. She feels that if he falls in the house when he is alone he could conceivably die of exposure, or seriously injure himself again. The medical response to this is to alert the social services and let them get on with it. What help could they provide? Mr. F.K. already has a home help three times a week and this support could be increased if necessary. Meals on wheels could be provided and the “tucking in” service mobilised so that he is checked each night by a qualified nurse. Alternatively he could be put on the list for sheltered housing. He is fully mobile (though a little slow), fully continent and not greatly demeaned. On the other hand if his condition is considered likely to deteriorate over the next year or so, pant IV accommodation might be a wiser option.

The ideal would be for him to stay at home and be helped to look after himself by his daughter and the support of the social services. Any home-aids which he may require in time could be provided.
Mr F.K. is at present still on Ward 12, four weeks after his admission. He is eager to know what has been discovered, although nobody has told him yet. His daughter has been informed about the suspected malignancy. His admission has involved the development and treatment of a urinary tract infection and a large degree of boredom. Everyone, especially the patient, will be glad when a decision about his future management is finally reached.