

DATA ON SIX CASES OF CEREBROVASCULAR ACCIDENT  
AFTER CANNULATION OF THE INTERNAL JUGULAR VEIN

Case	Carotid puncture	Hemi-paresis	Manual compression	Haematoma	Evidence of arteriosclerosis	Necropsy
1 (66, F) <sup>11</sup>	R	L	?	-	+	-
2 (57, M) <sup>13</sup>	R	L	(+)	+	++	+
3 (71, M) <sup>13</sup>	R	L	(+)	-	+	Alive
4 (74, F) <sup>12</sup>	R	L	+	(+)	++	+
5 (72, M) <sup>12</sup>	L	R	+	(+)	+	-
6 (70, F)	R	L	(+)	-	-	-

A 70-year-old, severely ill woman was admitted with a dynamic ileus due to abdominal cancer. There was no sensory or motor deficit and she had no evidence of cerebrovascular insufficiency. Carotid and peripheral artery pulses were normal bilaterally. The ECG showed sinus rhythm without ischaemic signs. Platelets and coagulation status were normal. During cannulation of the right internal jugular vein, the carotid artery was punctured and this was immediately recognised. The catheter was withdrawn and the region was gently compressed. The patient became cyanotic, flaccid, and finally unresponsive to stimuli with tonic deviation of the eyes to the right. Left-sided hemiparesis was diagnosed and dexamethasone, sodium bicarbonate, and dextran 40 were given via a catheter inserted into the femoral vein. There was no bradycardia or arrhythmia. Chest X-ray showed no abnormalities. The paresis improved within an hour but the patient did not fully recover and died 8 h later. Permission for necropsy was refused. Since there was no evidence of extrinsic haematoma compromising perfusion, hypotension, or vasovagal arrhythmia the precipitating event may have been laceration or dissection of the intima of the carotid artery, dislodgement of friable atheromatous plaques, or even application of manual pressure.

This case and five other published ones are summarised in the table. The mean age of the patients was 68 years and all but one had evidence of arteriosclerotic disease. Besides carotid artery puncture during cannulation, a feature common to all patients was the application of manual compression to avoid haematoma formation. Although there was no doubt about the temporal and local anatomical relations between the catheterisation and the manifestations of neurological injury the mechanism is unclear; only in patient 2 was a haematoma found to be compressing the common carotid artery.

More attention should be paid to this fatal complication. In non-emergency cases cerebrovascular insufficiency should be looked for, especially in elderly patients, before the catheter is inserted. Should the carotid artery be punctured awareness of the possibility of a cerebrovascular accident should help it to be recognised early so that treatment can be started promptly.

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### IN-VITRO FERTILISATION PREGNANCY WITH EARLY PROGESTAGEN SUPPORT

SIR,—On July 13, 1982, a healthy boy weighing 2340 g was born after the first successful in-vitro fertilisation pregnancy in Western Australia.

A 28-year-old woman had a 7 year history of infertility from inflammatory tubal disease. After tubal surgery, first one and then a second tubal ectopic pregnancy occurred with a salpingectomy undertaken on each occasion. At subsequent laparoscopy, absent fallopian tubes and non-patent tubal stumps were noted. During her treatment cycle, the patient was given clomiphene 150 mg on days 2–6 after her last menstrual period on Oct. 13, 1981. Intramuscular human chorionic gonadotropin (hCG) 5000 units was given on day 12, when an ultrasound confirmed a 2.1 cm follicle in the left ovary and features suggesting a dispersed follicle in the right ovary with evidence of some fluid in the pouch of Douglas. At laparoscopy on day 14, a mature follicle was aspirated on the left side, and a healthy

preovulatory oocyte was recovered. In addition a dispersed follicle was flushed and aspirated on the right side, and this also revealed a mature oocyte retained within. A few millilitres of fluid was aspirated from the pouch of Douglas revealing follicle debris, but no oocyte. The recovered oocytes were fertilised in 1 ml of Hams F10 solution containing 7.5% deactivated maternal serum.<sup>1</sup>

Fresh semen was collected from the husband at the time of laparoscopy and, after resuspension in Hams F10 solution, approximately 200 000 spermatozoa were added to each oocyte tube. At 20 h the oocytes were transferred in solution to petri dishes where the coronal coat was dissected off revealing single cell oocytes, each containing two visible pronuclei. Each was transferred to a tube containing Hams F10 solution with 15% deactivated maternal serum. At 44 h after oocyte recovery, the embryos were noted to have reached the three-cell and five-cell stages. Embryo transfer was carried out at 53 hours, via a polyamide catheter (Portex), when the embryos were at the four-cell and eight-cell stages. The less advanced embryo showed moderate fragmentation.

The patient was given medroxyprogesterone acetate 10 mg oral four times daily for 10 days beginning on the second day when fertilisation was confirmed. After 12 h of bed rest the patient was discharged. The medroxyprogesterone was stopped after 10 days and restarted 30 days after oocyte recovery when the pregnancy had been confirmed, but signs of threatened miscarriage appeared (bleeding and fall in basal temperature). Medroxyprogesterone was continued until 16 weeks' gestation by menstrual dates.

Fig. 1 shows the rising  $\beta$ -hCG levels with pregnancy diagnosed 13 days post-laparoscopy. Weekly serum oestradiol and progesterone levels were monitored, and fig. 2 discloses the interesting feature of steadily rising oestradiol with progesterone

1. Wood C, Trounson A, Leeton J, McKenzie-Talbot J, Buttery B, Webb J, Wood J, Jessup D. A clinical assessment of nine pregnancies obtained by in-vitro fertilisation and embryo transfer. *Fertil Steril* 1981; 35: 502–08.

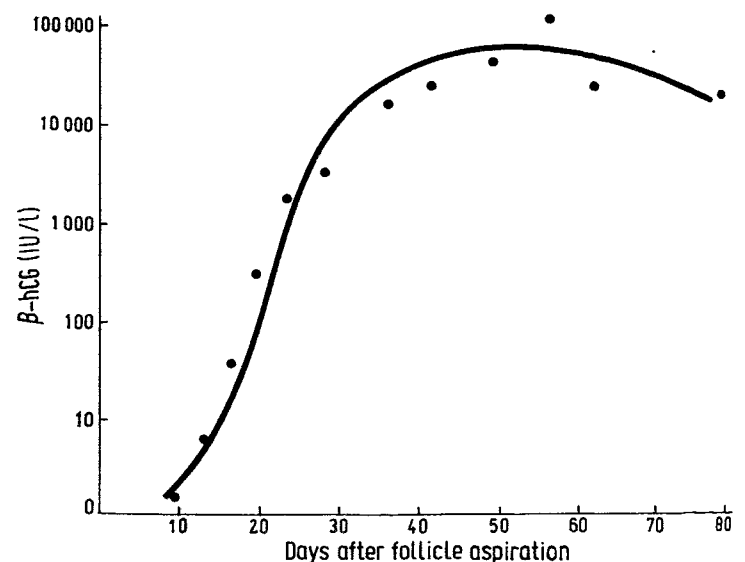


Fig. 1— $\beta$ -hCG concentrations.

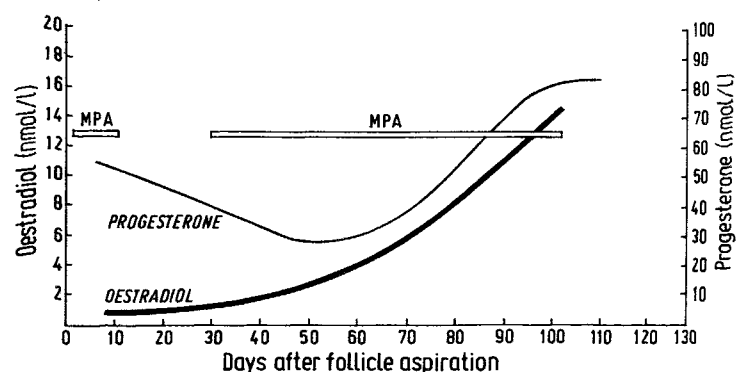


Fig. 2—Oestradiol and progesterone values. MPA = medroxyprogesterone acetate.

levels gradually falling, the lowest level being recorded around 9 weeks' gestation by menstrual dates. There was a gradual return to normal levels by 16 weeks' gestation. Ultrasound confirmed a gestational sac 4 weeks after embryo transfer, and was repeated at the 8, 12, 16, and 28 week stages. Ultrasound development was around the mean level for the various indices at each stage, although the abdominal girth was at the 10th percentile at 28 weeks. In the latter weeks of pregnancy, plasma oestriol measurements were always normal, but there were clinical features suggesting growth retardation (low weight gain, oligohydramnios, and small for dates). Induction by rupture of the membranes and intravenous oxytocin was done on July 13 and the fetal heart rate was monitored by a scalp electrode. As contractions became established deep decelerations led to early caesarean section under epidural anaesthesia. The infant was a healthy vigorous male at birth showing signs of intrauterine growth retardation. There are no abnormalities and he has established breastfeeding satisfactorily.

This pregnancy can be added to those from the few other teams who have reported successful in vitro fertilisations.<sup>2</sup> It happened during a pilot study with a budget of only about £8000 during which forty-two cases were treated. It is only in the last twenty-two that the rate of fertilisation rate of cleavage and morphological appearance of the embryos suggested that success was likely. In the series four "chemical" pregnancies were diagnosed by raised  $\beta$ -hCG levels, but none advanced to the stage of confirmation. Progesterone support was given to randomly selected cases. Medroxyprogesterone acetate was selected for its ease of administration and lack of reports of human embryopathy.<sup>3</sup> Its role in the above case during the luteal phase is uncertain, but we do suspect a contribution from progesterone support in those cases which subsequently display aberrant luteal phases, and we believe that medroxyprogesterone acetate combined with rest did prevent spontaneous abortion of this pregnancy during its early weeks. We are continuing further studies of this agent to assess its role in infertility management, support for threatened miscarriage, and a possible role in improving the embryo transfer success rate after in vitro fertilisation. It is of additional interest that an oocyte was recovered from a dispersed follicle and successfully fertilised. This has previously been reported.<sup>4</sup>

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### LEFT STELLECTOMY IN MANAGEMENT OF INTRACTABLE IDIOPATHIC CHEST PAIN IN PATIENT WITH CORONARY DISEASE

SIR,—A 52-year-old man presented in 1979 with severe prolonged retrosternal pain radiating to the centre of the back at T7. The electrocardiogram revealed an old transmural inferior wall infarction and non-specific ST-T wave changes. Admission to a coronary care unit was arranged and the pain was treated with narcotics. There was no biochemical or ECG evidence of acute myocardial infarction. This was the first of twenty-two similar admissions, over 23 months.

The patient had had a myocardial infarction in 1975 with post-

infarct stable angina. Anterior descending and right coronary artery saphenous vein grafts were constructed in June, 1975, and the patient remained pain-free for 30 months. In February, 1978, he was admitted with "unstable angina" and there were several further admissions for spontaneous central chest pain without ECG changes. In June, 1979, catheterisation studies confirmed graft patency. In the repeated episodes there were two pain patterns. One was an epigastric "heavy" sensation with back, left shoulder, and left elbow radiation, usually occurring during exertion. The other was a frequent and severe back pain beginning at the inferomedial border of the left scapula, radiating around the left mid-axillary area and on to the central retrosternal zone. This pain was not provoked by exertion but was exacerbated by emotional stress, and lasted as long as 5 days. The pain did not respond to nitroglycerin and could only be relieved by narcotics.

Hospital investigations were oriented towards myocardial infarction and, later, unstable angina, but ECGs recorded during pain episodes never revealed any deviation of the ST segment. In all early admissions, serial cardiac enzymes were normal. Chronic stable angina probably explained the first pain but was clearly not the source of the second type of pain, which required hospital admission. Treadmill testing provoked pain 1, with equivocal ST segment depression, and myocardial perfusion imaging with thallium-201 revealed exercise induced apical and posterior wall ischaemia. The second (and more troublesome) pain remained unexplained, despite extensive radiological and endoscopic investigations. Plain X-rays revealed degenerative disease in the cervical, but not the thoracic, spine; and myelography demonstrated minor filling defects at C4-C5 and L4-L5, but the thoracic region was normal.

The patient was anxious, as would be expected in chronic undiagnosed pain. The disabling pain continued after the patient's forced retirement: his preference was to return to work, which he achieved only after stellate ganglion surgery.

Psychotherapy, behaviour modification and anxiolytic agents were unsuccessful, and the pain continued, despite trials with many drugs oriented toward the cardiac, gastrointestinal, muscular, and psychological systems.

After a 2 year period of many investigations, consultations with several specialists, and countless drugs—all unhelpful—stellate ganglion blockade was tried. (Lignocaine blockade of T7 nerve roots, done during a pain episode produced anaesthesia in the appropriate dermatome but no relief from the back or chest pain.) Left stellate ganglion blockade led to the immediate disappearance of the second type of pain, and the appearance of a left Horner's syndrome. Blockade was repeated on twelve occasions and pain relief was achieved ten times—i.e., on every occasion when Horner's syndrome appeared, indicating successful blockade. Placebo injections produced no relief. Surgical extirpation of the left stellate ganglion was done in November, 1981, and the type 2 pain has never reappeared.

Because the patient had a history of coronary artery disease his physicians were most reluctant not to admit him to a coronary care unit. As successive admissions failed to yield a diagnosis, and despite the assurances of the psychiatrists to the contrary, a psychiatric label was affixed. Now, 9 months after stellate ganglion surgery the non-anginal pain has gone and low dose beta blockade controls the angina. The patient has exercised to the point of exhaustion (7 min) without pain and now has no symptoms and no arrhythmias.

Stellate ganglion block was recommended as a temporary measure in intractable angina long before we understood the physiology of angina and revascularisation surgery;<sup>1</sup> we used it as a last ditch measure on strictly empirical grounds, in the hope of ending a cycle of severe pain and repeated hospital admission. It is encouraging that, besides the clinical success, no adverse cardiac effects have emerged.

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