make general practitioners' contracts more work sensitive. Finally, there was a strong case for settling the new contract so that the profession would be free to concentrate on the NHS review and its potentially damaging effects on the health service.

Much of the doctors' anger has arisen because of a belief that the revised contract was a fait accompli. That is wrong. Although the negotiators agreed to commend the package to general practitioners, the Secretary of State was told that a final decision on agreement or rejection rested with the conference of representatives of local medical committees on 21 and 22 June. Dr Wilson and his colleagues were understandably upset that doctors and local medical committees had jumped to conclusions before assessing the detailed contents of the package or hearing his briefing on the background to the negotiations. Certainly, many GMSC members who had come prepared to bury the package returned to their constituencies persuaded that it was worth unwrapping.

Doctors have also criticised the seeming hastiness of the 4 May agreement. But the seeds of this contract were planted in 1980 on the initiative of the local medical committee conference, which approved a new charter for general practice. Repeated attempts by the GMSC to negotiate changes in general practitioners' contracts were thwarted by policies to contain public expenditure and by repeated government promises of a green paper on primary care. The promise eventually materialised in November 1987 as the white paper Promoting Better Health, negotiations on which started in March 1988 and culminated in February 1989 with Kenneth Clarke's new contract. So when the two sides met on 4 May for what was expected to be the first of several ministerial level meetings each side was thoroughly grounded on the other side's views and aspirations. Unexpected progress was made—perhaps influenced by outside political events—and both sides took the opportunity to put a package together.

An understanding of the details and background of the package are essential if doctors are to reach a constructive decision in June. Doctors should recognise, too, the political importance of resolving the contract dispute because there are some highly damaging prospects for general practice in the white paper Working for Patients. The profession needs a clear field of fire to win that battle: Dr Wilson and his colleagues have provided an opportunity to ensure this.

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Genetic factors in hyperactivity

Account for about half of the explainable variance

Whereas parents use "hyperactivity" as a label for a broad range of childhood behaviours, including normal exuberance and frequent night wakenings, child psychiatrists employ the term to refer to restlessness and inattentiveness that are inappropriate for the child's age. Hyperactivity in this restricted sense is commonly accompanied by other cognitive, educational, and psychiatric handicaps and may affect about 1% of British primary school children.

Family, adoption, and twin studies all suggest that genetic factors are important in hyperactivity. Thus one large study found that maternal half siblings who grew up together were considerably less alike in measures of hyperactivity than full siblings. Another study of siblings who had been fostered separately found that concordance for hyperactivity was much lower for half siblings than for full siblings. When compared with the parents of children who were not hyperactive the biological parents of hyperactive children were more likely to have attentional problems in adult life, and they were also more likely (as judged by parental recall) to have been hyperactive themselves as children.

Adoption studies suggest that these cross generational continuities reflect genetic rather than cultural transmission. Thus no excess of attentional problems or retrospectively diagnosed hyperactivity has been found in the adoptive parents of hyperactive children who were adopted when they were very young. Twin studies also point to a genetic contribution: monozygotic twins are more alike than dizygotic twins of the same sex in measures of hyperactivity and inattention. Even when allowance has been made for parents' and teachers' tendencies to exaggerate the similarity of identical twins or minimise the similarity of non-identical twins, genetic factors account for about half of the explainable variance in hyperactivity. As the remaining half is presumably environmental this finding emphasises that both genetic and environmental effects are important.

Little is known about how hyperactivity is inherited, although some evidence favours polygenic transmission. Different modes of inheritance may operate in different families as hyperactivity is probably caused by many different genetic disorders. Among the mentally retarded, for instance, hyperactivity is associated with several genetic conditions, including tuberous sclerosis, mucopolysaccharidoses, and untreated phenylketonuria. Hyperactivity without mental retardation may also be genetically heterogeneous. For example, some hyperactivity in non-retarded children may be caused by the gene (or genes) responsible for schizophrenia—thereby explaining why schizophrenia is commonly preceded by childhood inattentiveness and why the children of parents with schizophrenia have a particularly high rate of attentional problems.

The knowledge that genes play an important part in hyperactivity is relevant to clinical practice. When faced with a hyperactive child doctors should remember that the parents' child rearing techniques are not necessarily to blame. Indeed, the parents may well deserve credit for how well they have coped with the child's severe and often exasperating handicap.

As is well shown by phenylketonuria, the fact that a disorder has a genetic component is not necessarily a cause for therapeutic pessimism. When more is known about which psychological factors influence hyperactivity it will be possible to look for clinically important interactions between genes and the environment. If psychosocial factors either amplify or suppress a genetic predisposition to hyperactivity
this will help treatment and prevention. The new tools of molecular genetics open a second promising avenue for future research.\textsuperscript{15} Relevant genes may be located and their products identified, leading possibly to improvements in biological treatments. For example, knowing that a genetic predisposition to hyperactivity was mediated by a defect in a neurotransmitter receptor might aid pharmacotherapy.

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Fetal uropathy

Conservative management is best

Ultrasoundography permits the prenatal diagnosis of diseases of the fetal urinary tract. The abnormalities fall into two main categories: persistent dilatation of part or all of the urinary tract, suggesting possible obstruction; and a miscellaneous group consisting of either cystic changes, increased echogenicity, or reduced renal parenchyma. Obstructive lesions are diagnosed more commonly prenatally than non-obstructive lesions. Diagnosing the non-obstructive lesions is less important because they are usually either irremediable or unlikely to damage the kidneys before birth. The main aim of prenatal diagnosis is thus to spot obstructive lesions.

The estimated incidence of fetal uropathy varies widely,\textsuperscript{1,2} but the prospective study of over 6000 pregnancies reported on p 1417 gives an incidence of almost eight in every 1000 pregnancies. Forty two cases were diagnosed prenatally, and a further seven infants whose ultrasonographic scans showed no abnormalities were detected later when they presented with symptoms.

Fetal kidneys are already secreting urine towards the end of the first trimester, and severe early obstruction causes not only hydrenephrosis but also dysplasia.\textsuperscript{3} The removal of metabolites by the placenta may, however, ensure survival to term—even in fetuses with bilateral renal disease. In later pregnancy fetal urine is an important source of amniotic fluid, and deficient renal function may lead to oligohydramnios, resulting in compression of the limbs and face and pulmonary hypoplasia.\textsuperscript{4} Relieving the obstruction before birth will be effective only if the kidneys have not already been irreparably damaged,\textsuperscript{5} and two further papers (p 1419 and p 1421) report high fetal and early neonatal mortalities from fetal uropathy. The deaths resulted mostly from bilateral obstructive nephropathy or from primary defects such as agenesis or polycystic disease; but some resulted from lethal extrarenal defects, including chromosomal abnormalities.\textsuperscript{6} Pulmonary hypoplasia was the cause of death in the first patient to be given a prenatal operation,\textsuperscript{7} and accounted for nine tenths of deaths in the report of the international fetal surgery registry.\textsuperscript{8} Overall, between a half and two thirds of fetuses with uropathy die.\textsuperscript{9,10} Intrauterine surgery requires considerable technical skill, and the international registry showed that from two to seven attempts were made at inserting vesicoamniotic shunts in nine tenths of cases.\textsuperscript{11,12} Furthermore, the procedure itself causes death in one in 20 cases and complications in almost half.\textsuperscript{11,12}

The findings published today agree with those in previous reports\textsuperscript{11,12} in providing little support for intrauterine operations, and attention should be focused on conservative management.\textsuperscript{11} Ultrasonographic examination between 17 and 20 weeks' gestation will show most lethal urological abnormalities, but less serious defects may not be detected until 28 weeks or later.\textsuperscript{13,14} Unilateral uropathy does not kill, and pregnancy should always be allowed to proceed to term. Severe oligohydramnios occurring before mid-pregnancy always kills because of pulmonary hypoplasia or renal failure.\textsuperscript{15} Other serious extrarenal defects may be detected ultrasonographically, but karyotyping is needed in fetuses with bilateral uropathy because of its frequent association with chromosomal abnormalities.\textsuperscript{16} Increased sodium, chloride, and osmolar concentrations in fetal urine obtained by percutaneous bladder aspiration consistently predicted an unfavourable outcome in one series,\textsuperscript{17} and this procedure may be of additional value when considering the options of termination of pregnancy, continuation to term, or induced labour if, exceptionally, the risks to the kidneys from continuing pregnancy are judged to outweigh those of prematurity.

The sensitivity of prenatal ultrasonography in detecting uropathy is now high,\textsuperscript{18} although one difficulty is distinguishing between renal cysts and obstructed calices.\textsuperscript{18} But the rate of false positive results is also high—50% in one of the series published today. Such errors of interpretation cause considerable parental anxiety, and counselling needs to be both sensitive and informed.\textsuperscript{11,13} Transient urinary tract dilatation is often seen in mid-pregnancy, and we do not yet know if this is a manifestation of undiagnosed vesicoureteric reflex. Five of the seven missed cases in the series reported on p 1419 were found to have reflex. The findings of the Birmingham Reflex Study Group\textsuperscript{17} support the contention that the renal scarring associated with reflex begins at the time of the first urinary infection.\textsuperscript{18} Antenatal screening therefore provides an opportunity for preventing reflex nephropathy if